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VOLUME XI—1941-1942

ISSUED QUARTERLY

Single Numbers, 7s. 6d. Annual Subscription, £1 10s.

SYDNEY
BUTTERWORTH & CO. (AUSTRALIA) LTD.
(Incorporated in England)
6-8 O'Connell Street

Melbourne, Vic.: Clarke Buildings, 430 Bourke Street.
Wellington, N.Z.: 49-51 Ballance Street.
Auckland, N.Z.: 35 High Street.
London: Butterworth & Co. (Publishers) Ltd., Bell Yard, Temple Bar.
Calcutta / Bombay } Butterworth & Co. (India) Ltd.
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The Australian and New Zealand Journal of Surgery

JULY, 1941.

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Exchange journals should be addressed to the Honorary Librarian, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1, Victoria, Australia.

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THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

Registered at the General Post Office, Melbourne, for transmission by post as a periodical.

VOL. XI. JULY, 1941. No. 1.

CEREBRAL ABSCESS: A REVIEW OF THIRTY CASES IN A GENERAL HOSPITAL.[1]

By A. E. COATES,
Melbourne.

THIS review of cases of brain abscess is necessarily limited, for the reason that the hospital concerned is a general hospital for adults in which only a limited number of special cases are treated. The nose, ear and throat department is a consulting one for general cases and consequently there is not the same amount of severe disease of that specialty as would be found in a special hospital. On the other hand, the occurrence of brain abscess in association with general disease is of interest, and while it is not confidently expected that much will be gained by a consideration of this group of cases, it is possible that increasing interest in this terrible complication may lead to earlier diagnosis and more successful treatment than has hitherto been obtained. The terms primary and secondary abscesses are often applied in our records; such terms are meaningless. Every abscess is secondary to some preexisting lesion, whether it be a penetrating wound, a suppurating mastoid, or a lung infection. It is the method of spread to the brain tissues which may distinguish this type of case. Therefore I propose to classify the cases according to the probable source of infection.

Reading carefully through the histories of these patients, some of whom I saw myself, and comparing the post-mortem findings, one cannot escape the conclusion that a few of these patients could possibly have been saved by timely operation. In most cases diagnosis was uncertain, but in those in which it was almost conclusive, the treatment was perfunctory.

MORTALITY.

I have excluded from this series consideration of two patients of my own who recovered. One had a large extradural abscess, a metastasis from old osteomyelitis, puerperal pyæmia, and what was probably a small intracerebral abscess. The other had a fungous infection of an intracerebral hæmatoma. Drainage of the infected site by corrugated rubber led to recovery in eight weeks. Of the other 30 patients, two only recovered—that is, 6·6%. One patient was discharged and lost sight of, but he almost certainly died later. The mortality then in this small series of proven localized intracerebral collections of pus is 93·3%.

The accompanying table indicates the cause of the abscess in thirty cases.

[1] Accepted for publication on July 18, 1939.

TABLE I.

Condition.	Number of Cases.
Frontal sinus	1
Osteomyelitis of the skull	2
Otitis media	7
Lung infection	11
Penetrating wounds with fracture of the skull	3
General infections[1]	6

[1] Included among these was one case of cancer of the liver and one of pneumo-meningitis.

Two patients recovered. One had a penetrating shrapnel wound of the cerebellum; the abscess was excised with the shrapnel fifteen years later. The other suffered from *otitis media* and had a temporo-sphenoidal abscess.

The post-mortem records show that six of the patients had multiple (two, three or more) abscesses. Each of the remaining 24 patients had a solitary abscess. This is an important point. In five of the patients in whom the cerebral abscess was secondary to pulmonary suppuration, the abscess was solitary.

The history of symptoms and signs varied between weeks and six months. Five of the abscesses were described as being very large. One was in the basal ganglia. The occurrence of abscess in relatively silent areas (nine in the frontal and four in the temporal lobes) may account for the absence of localizing signs. That twenty-four of the patients suffered from a single abscess encourages me to believe that earlier and more accurate diagnosis might have brought some of them to successful operation. Signs of meningeal irritation were noted in ten cases. Probably such signs were present and unobserved in a greater number. In eight of the ten cases the cerebro-spinal fluid was clear; in two only were organisms found. In four cases with sterile cerebro-spinal fluid a marked increase in cells was noted.

In my own cases I have been impressed with the inconclusive evidence afforded by examination of the cerebro-spinal fluid. Meningismus, occipital pain, increasing cerebro-spinal fluid pressure and high cell content in a sterile fluid would lead one to suspect the presence of a cerebral abscess.

I do not propose to discuss symptomatology beyond suggesting more constant and repeated neurological examination, especially repeated perimetry. The sequence of events in a case of suppuration in the air passages, mastoid cells and sinuses, or the development of cerebral symptoms in a case of lung or bone abscess, should prompt the attendant physician or surgeon to seek the neurologist's aid. Neurosurgical technique has developed today to such an extent that certain special investigations may aid in the diagnosis. It is widely held that if an abscess has developed, the longer it remains, the more likely it is to be amenable to surgical attack. This is only a half-truth. Certain it is that a general inflammation or phlegmon following a penetrating wound or suppurating mastoid is not a surgical problem, except in so far as the provision of efficient drainage to the local condition is urgently necessary. It is comparable with general peritonitis in the presence of a gangrenous appendix. There is a period during the patient's fight with the offending organism when efficient barriers are being erected, when an abscess wall is forming and localization is taking place. But there is a good rule in other areas which is worth bearing in mind in cerebral infections, and that is that pus should not be allowed to accumulate under pressure. The policy of waiting can be overdone.

In one of the cases in this series I suspected that an abscess was present in the right frontal area. I watched the patient for a few days, and arranged to operate on my next operating day. The poor girl died the night before the planned operation, and post-mortem examination disclosed a solitary large abscess, thick-walled, which could easily have been relieved and subsequently excised.

A decompression should have been done in that case, allowing the abscess to bulge outwards, relieving general brain pressure, and then the emptying of the cavity and its ultimate enucleation would have been possible. Vincent, of Paris, has been advocating this treatment and his results speak for themselves.

While one is aware of the great difficulties in diagnosis of cerebral abscess, while fever may be absent, fundi normal and meningeal changes few, one cannot avoid the conclusion that a policy of despair is adopted by many practitioners in dealing with these cases. I plead for the earlier help of neurologist or neurosurgeon. The result of special investigation, such as a plain skiagram showing a gas bubble in the abscess, displacement of pineal body or an area of petrositis, may lead to the diagnosis and localization of the abscess.

Localizing information may be obtained by ventriculography. I have been diffident about performing this operation in case of abscess, but a study of Vincent's work convinces me that it may be done without increase of the mortality rate. The terrific risk (in our case 90% or more) of fatality if the "wait and see" policy is adopted, cannot be increased very much. Furthermore, the ventricles may be tapped in a site away from the suspected abscess and the risk of rupture of an abscess as a result of the lowering of the pressure seems to be surprisingly slight. These remarks apply to hemispherical abscesses, not to cerebellar collections. In the latter, a suboccipital exploration would reveal the side affected.

From a study of autopsy material and a review of the cases presented, I am convinced that a proportion of these patients may be brought to operation with some hope at least of the purulent collection being attacked. As neurosurgical work becomes more widely recognized, it is probable that fewer of these patients will be moribund on transfer to our clinics. Furthermore, we must be prepared to explore the brain more frequently in suspected cases. Cairns emphasizes four points which preclude the presence of an abscess at any rate in the immediate vicinity of the decompression. They are: (i) the presence of free pus in the subarachnoid space, (ii) an abundant flow of clear cerebro-spinal fluid, (iii) the recovery of more than one cubic centimetre on a ventricular tap, (iv) the normal appearance of the brain at the site of exploration.

It is desirable to approach the abscess through a clean field—that is, to seal off the old track, the primary sources of infection. Local anæsthesia may be employed as for tumour cases, or the inhalant which is thought most desirable may be used. If a temporo-sphenoidal abscess is suspected, a subtemporal decompression may be done; if a frontal abscess is suspected, an osteoplastic flap may be cut. In any case, avoidance of heavy trauma, such as caused by a mallet or chisel, is desirable. My sole experience of the bone flap operation is confined to the case of a fungous infection that I have mentioned. The bone flap had lifted after the first operation; at a second stage it was easy to open the bulging collection and drain it. Vincent's method of cutting a bone flap without an opening of the dura allows the abscess to project. Sealing the dura to the brain with the diathermy permits the safe introduction of a cannula and the withdrawal of pus. Closure of the wound and subsequent tapping and bacteriological and cytological study enable the surgeon to determine when the abscess is ripe

for enucleation. This method may be varied. Marsupialization of the lining of the abscess as advised by Horrax and suture of the capsule to the skin may succeed, or "uncapping" the abscess as advocated by Kahn may be effective. In this method, a flap having been cut, the abscess is discovered by tapping, then a small circular hole one and a half inches in diameter is cut in the flap and the abscess is allowed to bulge through the gap. Loose iodoform gauze is placed over the œdematous brain. Sucking away the soft covering brain three days later, Kahn succeeds in removing the abscess, capsule and all. This treatment would appear to be applicable in cases of deep abscess. Dandy relies on simple aspiration. Certainly superficial abscesses are accessible by this method. Coleman prefers aspiration followed by the introduction of a soft rubber catheter. His results are comparable with those of MacEwan.

The difference of opinion among neurosurgeons as to the best method of dealing with cerebral abscesses indicates that the matter is still *sub judice*. If we recall the history of the advances of surgery in the treatment of purulent collections in other cavities—for example, the abdomen—it is safe to predict that during the next few years the pooled experience of surgeons will enable a more definite pronouncement to be made. Probably the various methods advocated have a place in treatment of abscesses in different stages and in differing sites.

A surgical repertoire is as necessary in the treatment of this condition as in dealing with lesions elsewhere in the body. My disquieting experiences of the disease would indicate that accurate localization of the abscess is a first essential. Secondly, a decompression, preferably by osteoplastic flap, is urgently required, allowing more room for the swollen brain and the abscess to direct itself to a safe exit. The further treatment of the abscess may then be considered at leisure. Vincent has shown that the dura need not be opened and yet a substantial bulge will occur.

SUMMARY.

A review of thirty cases of cerebral abscess is made. The high mortality is noted. A few suggestions for the improvement of diagnosis and treatment are offered. A plea is entered for close cooperation between the general and special branches of the profession so that a chance of recovery may not be withheld from those who would otherwise die. The fully developed intracranial abscess presents a problem requiring the diagnostic and therapeutic armamentarium of the neurosurgical service.

SURGICAL TREATMENT OF HÆMATEMESIS.[1]

By G. R. A. Syme,
Melbourne.

Hæmatemesis is a condition which is usually treated very successfully by medical methods, but there is a certain proportion of cases, probably about 10%, in which the hæmorrhage is coming from a large vessel, and these cases present the same problem as hæmorrhage from a large vessel in a limb—that is, the vessel must be ligated or else the patient will die. Nobody would treat a limb hæmorrhage by medical means, so why attempt it in the stomach, which is accessible to surgery? The answer usually given is that most hæmorrhages from the stomach are from small vessels and stop spontaneously; with this I agree, but some do not, and it is our duty to attempt to divide the cases into two classes, medical and surgical.

I have operated in only 15 such cases, and statistics based on so small a number are worthless; but the results of studying them, particularly the five in which death occurred, are quite instructive.

Indications for Surgery.

Every surgeon has a list of indications for operation in peptic ulcer which is now almost standardized—that is, perforation, obstruction, possibility of cancer, and failure to respond to medical treatment. Should bleeding occur in such a case, there is an added indication to operate urgently, and there is certainly no indication to postpone surgery.

Unfortunately, few of our cases are in this group, so we have had to lay down some rules for guidance in the remainder. Firstly, it has been found that death is rare in patients under the age of forty years, possibly because of the greater retractile power of arteries in younger persons, so we are very loath to operate on any patient under forty.

Secondly, as Gordon-Taylor has shown so convincingly, a history of a previous hæmorrhage greatly increases the probability of death if medical treatment only is maintained, so it becomes a strong factor in deciding on operation. Thirdly, a good ulcer history of long duration is usually required, but is not an absolute necessity.

It is not contended that by the observation of these rules no unnecessary operations will be done or that no patients who are amenable to surgery will be refused operation and ultimately die, but the number will be reduced to a minimum.

The Time to Operate.

Because of the frightening appearance of the condition, every patient with hæmatemesis is very soon seen by a medical man, and this is the time at which one should decide whether or not surgery is indicated and, having made a decision, to act accordingly without unnecessary delay. Do not say, "If he bleeds again I will operate"; and certainly, once having decided that the case is a medical one, and having refused operation, do not let yourself be persuaded at the eleventh hour, when medical treatment is apparently failing, to try a last hope exploration, for this will certainly kill the patient while he still might have a faint chance if left to medical means.

[1] Accepted for publication on April 19, 1939.

A patient should obviously be allowed to recover from shock before he is operated on, and many will be much benefited by a blood transfusion, but most will be fit for operation in from twelve to eighteen hours. Further delay may allow a second hæmorrhage to occur and consequently increase the risk of unsuccessful operation.

THE OPERATION.

Local anæsthesia, with a field block followed by a splanchnic injection, was used in the majority of my cases and was usually efficient, but in the more difficult ones the need for a deeper anæsthesia was felt and a high spinal anæthesia with "Percaine" was more effective.

A mid-line incision was used at first, but with local anæsthesia a transverse incision gave a better exposure and less retraction was required.

When operation is done, as advocated, soon after the first hæmorrhage, the surgeon may deal with the ulcer by his usual routine, provided that he treats the bleeding point by ligation, compression or excision. Sometimes in spite of the above indications and in spite of gastrotomy, duodenotomy and bimanual palpation with the ungloved hand, the ulcer will still be impalpable, and in these cases a subtotal gastric resection will be necessary. The importance of this statement was not realized until two patients after fruitless explorations had died of further hæmorrhages, and at post-mortem examination a small subacute ulcer had been discovered on the posterior wall of the stomach which even at this examination was quite impalpable.

REASONS FOR FAILURE.

Why are all the published results of the surgical treatment of bleeding ulcer, except those of Finsterer, so depressing? The answer is, even allowing for his excellent technique, that he is the only surgeon who operates early.

Thus delay in operating is the most important factor in failure; and although it was thought at first that we had in continuous massive blood transfusion a method which would allow us to delay operation, we have now come to the conclusion that something else besides blood is necessary, for even though the hæmoglobin value of patients who have had severe hæmorrhages is maintained above 75%, they show no tendency for either their abdominal or gastric incisions to heal. What it is that is missing we do not know, but we call it the "unknown factor"; probably it is vitamin.[1] Two of these patients after a satisfactory ligation have lived for a fortnight after operation with a hæmoglobin value over 75%; at the end of that time the abdominal wound showed no sign of healing and in spite of a usually satisfactory suture technique, the gastric anastomosis broke down, fistulæ and perigastric abscesses were found; also a large area of acute gastric erosion was present.

A second factor, which has been stressed by Hurst, is the fact that many of these cases present extremely difficult technical problems, and the prognosis would be bad in any circumstances.

A third factor is the impalpable ulcer which can be overcome only by doing a partial gastrectomy.

CONCLUSIONS.

1. Surgery is only required in about 10% of cases of hæmatemesis.
2. If operation is performed it must be done early if it is to succeed.
3. The operation must include direct attack on the bleeding point.
4. If the ulcer is impalpable, gastrectomy must be performed.

[1] Since this paper was delivered the "unknown factor" has been shown to be "hypoproteinæmia".

HYDATID DISEASE OF THE KIDNEY.[1]

By E. R. Reay,
Christchurch, New Zealand.

Hydatid disease is almost confined to the wool-growing countries. Not uncommon, it is a menace to the workers in the sheep industry and is productive of much human disability. The life cycle of the parasite has long since been discovered. Preventive measures to decrease the incidence of infestation have followed.

The hydatid worm (*Tænia echinococcus granulosus*) inhabits the small intestine of the dog. Each small worm at its due season discharges countless ova which are passed in the fæces of the dog. The ova are ingested in the contaminated food or water of the intermediate host. In New Zealand this is usually the sheep, which harbours the cystic stage of the parasite. As dogs frequently feed on the offal of infested herbivora, the life cycle of the parasite is completed.

The Development of the Typical Univesicular Parasite.

Occasionally man features as the intermediate host, and is the victim of cystic infestation. Following ingestion, the ovum lodges in the stomach. The chitinous shell of the ovum is dissolved in the gastric juices. There is released a cellular embryo with six hooklets by means of which an anchorage is established in the mucosa. Boring through the wall of the upper alimentary canal, the embryo reaches the portal circulation and the liver. In the narrow reaches of the hepatic capillaries it is usually arrested. If it passes beyond this barrier it still has to negotiate the pulmonary capillary system before reaching the general circulation.

Renal lodgment is uncommon and comprises but 2% of all cases. It may be preceded by infestation of the liver or lungs, but in the cases seen clinically the cyst is often primary and single. The embryo lodges in the renal cortex. It becomes surrounded by densely packed endothelial cells. If it is not overcome by this defensive reaction of the tissues, the solid embryo becomes vesiculated. As development proceeds, three definite layers may be distinguished in the vesicle (Figure I): (i) an inner nucleated lining membrane, the germinal layer; (ii) an outer laminated membrane; (iii) a surrounding fibrous adventitious capsule. This adventitia arises from cellular reaction in the surrounding tissues of the host. It is continuous with the interstitial tissue of the renal cortex. It closely resembles the fibroblastic reaction enclosing a tubercle.

The interior of the cyst is filled with fluid secreted by the germinal layer. The steady production of hydatid fluid causes growth of the cyst by continuous expansion. Expansion is symmetrical, except where an area of lessened resistance be encountered. Such a weakened area is the renal capsule. Cysts may protrude beneath the capsule to reach large proportions (Figure II). Other zones of weakness are the urinary channels. It is common for a portion of a cyst wall to prolapse into a calyx.

Follicular growths appear on the inner aspect of the germinal layer. From these the brood capsules develop, which in turn contain the scolices. When ingested by the final host, the scolices give rise to the mature worm.

[1] Accepted for publication on February 14, 1940.

The laminated membrane has separate and distinct functions. (i) It protects the developing scolices. (ii) Its osmotic properties allow the parasite to obtain nutriment from the host. (iii) It acts as a barrier preventing the entrance of noxious agents into the cyst. It limits the absorption by the host of products elaborated by the parasite. (iv) The membrane is highly elastic. When ruptured,

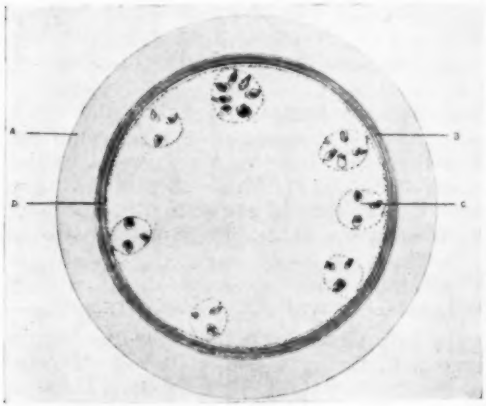

FIGURE I. Diagram of hydatid vesicle (after Dew). A = adventitia; B = laminated membrane; C = brood capsule with scolices; D = germinal layer.

FIGURE II. Closed cyst, showing early extrarenal protrusion.

FIGURE III. Latent cyst with a thickened calcified adventitia.

FIGURE IV. Plain skiagram of cyst shown in Figure III. Note the circular area of calcification.

it tends to turn inside out, thus ensuring the dissemination of the contents of the sac.

The hydatid fluid secreted by the germinal layer protects and nourishes the developing scolices. It contains certain specific proteins.

FIGURE V. Pseudo-closed cyst projecting into a calyx with absorption of the exposed adventitia; formation of daughter cysts.

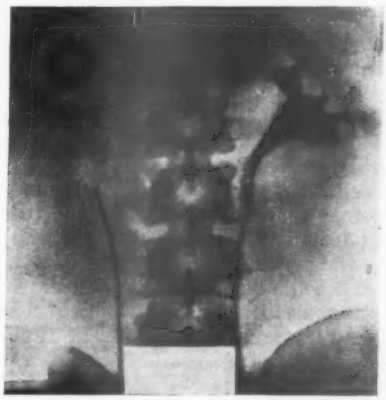

FIGURE VI. Pyelogram of cyst shown in Figure V; amputation of the inferior calyx.

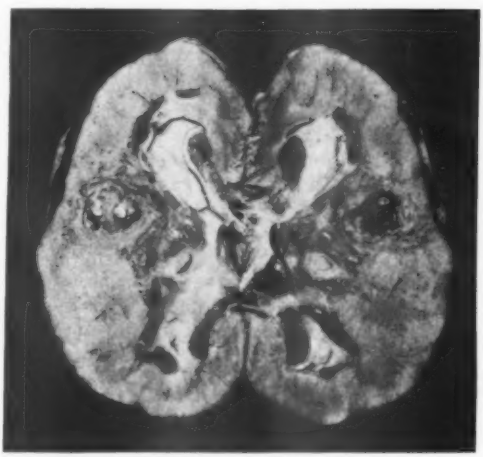

FIGURE VII. Cyst opening into the middle calyx; an unusual situation for a cyst, with early rupture.

FIGURE VIII. Primary cyst in the upper pole. Secondary implantation cyst in a dependent calyx. (After Begg.)

Sequelæ of Cyst Formation.

This shortly is the process of development of the typical univesicular parasite. It may live on, actively secreting fluid and expanding in the

tissues of the host. However, life may not be so placid in the busy economy of the human body. Constant warfare is waged between the parasite on attack and the tissues on defence. The defence forces may prevail. The cyst may also be subjected to traumatic or toxic influences. One of the following events may occur.

Latency.—Owing to an excessive tissue reaction, the adventitia may become so thickened and fibrosed that nutrition by osmosis is curtailed. Degenerative calcareous changes may occur in the wall of the cyst (Figures III and IV). Calcification may be circumferential or irregularly disposed in the wall of the cyst. The parasite lapses into a sluggish condition, inactive, but still alive. Calcification of the adventitia does not signify actual death of the parasite.

The Development of Daughter Cysts.—Daughter cysts are replicas of the mother cyst. They may float freely in the fluid, or may be packed so tightly as to render the sac almost a solid tumour. Their formation is the expression of a defensive reaction in response to some threat to the normal evolution of the parasite. Trauma, by detaching portions of the germinal layer, may initiate the process. Involvement of a urinary channel may lead to absorption of the exposed adventitia. The weakened laminated membrane in contact with the urine may allow the entrance of noxious substances into the cyst. There follows a reactionary formation of daughter cysts. They are rarely absent in renal cysts (Figures V and VI).

Death of the Parasite.—If an injury is so severe that no defensive reaction is possible, the laminated membrane retracts into a crumpled mass. The fluid is absorbed, the cyst shrinks and the parasite dies. Death may also occur from marked degenerative changes in the adventitia. Caseation takes place and the resemblance to a chronic tuberculous focus may be close.

Rupture of the Cyst.—Rupture occurs usually when a portion of the cyst wall encroaches upon the cavity of a natural channel. The soft tissues of a pyramid are easily absorbed by quiet pressure necrosis. The epithelial lining of a calyx is more resistant. As the cyst bulges into a calyx, the adventitia of the protruding portion becomes absorbed with the adjacent epithelial wall. The exposed laminated membrane may later rupture with total or partial emptying of the cyst (Figure VII). Rarely does the sac completely empty its contents. Usually some fluid is lost and the laminated membrane collapses and breaks up. It lies with a mass of daughter cysts in a ragged cavity bounded by the adventitia. Any of the constituent elements of the cyst may be found in the urine—membrane, daughter cysts, scolices or hooklets. The opening often becomes plugged with hydatid material. This process of fractional emptying may continue at long or short intervals over a period of years. The patient suffers attacks of renal colic from the passage of hydatid débris. One result of the rupture of an upper polar cyst may be the implantation of germ elements (scolices or daughter cysts) in a dependent calyx of the lower pole. A secondary cyst may quietly form. The walls of the calyx form the adventitia of the new cyst (Figures VIII, IX and X).

Suppuration within the Cyst.—The intact laminated membrane is impervious to organisms. Rupture is an essential preliminary to infection. The organisms commonly found belong to the *Bacillus coli* group. The adventitia is converted into a pyogenic membrane exuding pus. The tension of the purulent fluid is seldom high, and absorption is not marked. Infection rarely extends beyond the fibrous walls of the limiting adventitia.

The description of this series of events gives a prospect of the varied pathological states that may be encountered. They may be summarized (see Figure XI) as follows: (*a*) The closed cyst, with all layers intact, expanding

slowly in the renal substance. (*b*) The pseudo-closed cyst protruding into a calyx. (The exposed adventitia has been absorbed, leaving a potential space between the laminated membrane and the remaining adventitia.) (*c*) The open cyst, where permanently or intermittently there is a communication between the sac and the urinary channels. (*d*) The suppurating open cyst. (*e*) Secondary implantation cyst in a calyx.

General Effects of Hydatid Infestation.

The presence of even large cysts is compatible with good health. This is a feature of diagnostic importance. Cachexia may be present in cases of multiple infestation or when infection exists.

Definite specific effects may be produced by the absorption of proteins contained in hydatid fluid. These effects are lessened by the presence of a dense adventitia limiting absorption, and by inactivity of the parasite itself. Absorption is increased by intracystic activity, and especially by rupture. The following systemic manifestations may occur.

Eosinophilia.—Eosinophilia is commonly present, and especially during the early stages of development, and following leakage of fluid from the cyst.

The Elaboration of Antibodies.—Antibodies may form in the blood in response to absorption of hydatid antigen.

Sensitization.—The patient develops a state of sensitization to the antigen. The presence in the blood of antibodies and the sensitization of the patient to hydatid protein form the basis of the routine laboratory tests.

The complement fixation test depends on the presence of specific antibodies in the serum of patients who have absorbed hydatid antigen. So little antigen is absorbed from inactive or uncomplicated cysts that the result of the test may be negative. Interference with the parasite, such as operation, rupture or recent infection, increases the output of antigen. The result of the test will be strongly positive. Before operation the complement fixation test gives a positive reaction in approximately 80% of cases. The result should be negative within a year of successful operation. A strongly positive reaction after this period would indicate the presence of another cyst.

The Casoni test depends on the local reaction produced by the intradermal injection of fresh hydatid fluid in a sensitized patient. Unlike the complement fixation, this reaction may persist for twenty years after the parasite has been successfully eradicated. The test is of greatest value in simple uncomplicated cysts when the result of the companion test is often negative. The two are a valuable aid. Combined with clinical and radiological methods, they should secure the diagnosis in a high percentage of cases.

HYDATID ANAPHYLAXIS.

In a sensitized patient toxic manifestations may follow spilling of hydatid fluid at operation or after rupture. The hydatid rash (irregular urticarial wheals, with local or general erythema) may appear. Irregular fever may accompany dyspnœa and cyanosis. Profound anaphylactic shock may have a fatal termination. These effects would prompt the following operative precautions.

1. Careful aspiration and packing prevent soiling of the tissues with hydatid fluid during the evacuation of a cyst.

2. General anæsthesia is preferable at operation, as it usually abolishes the anaphylactic state.

3. It is wise to remember that a patient may be highly sensitized some fourteen days after rupture of a cyst. Operation may be deferred, as it is seldom a matter of urgency.

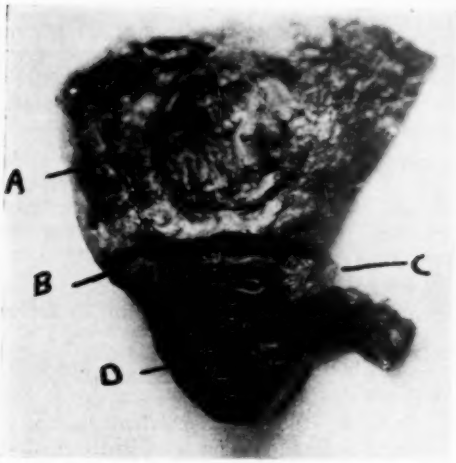

FIGURE IX. A = suppurating cyst of the upper pole. B = the opening into a calyx down which membrane C is passing to the pelvis. D = implantation cyst in the lower calyx.

FIGURE X. Pyelogram of cyst shown in Figure IX; calyces filled with hydatid débris.

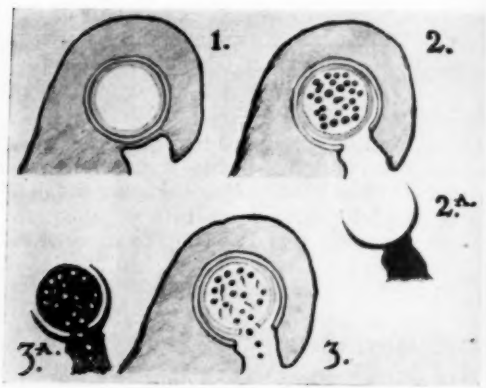

FIGURE XI. Diagram of 1 closed, 2 pseudo-closed, 3 open cysts. 2A and 3A are the corresponding pyelographic appearances of the affected calyces.

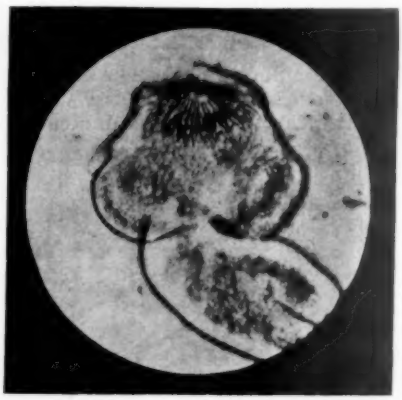

FIGURE XII. The scolex with its row of hooklets.

4. If a toxic reaction does occur, the administration of adrenaline may be useful.

SYMPTOMATOLOGY.

The closed kidney cyst, even when of large size, is often without symptoms. A large tumour may be discovered accidentally in a person in good health.

There may be obscure symptoms from pressure on abdominal organs or on a lung base.

In a personal case a cyst had been evacuated from the liver some years before the patient was seen. The man, an elderly farmer, had persistent jaundice and heart

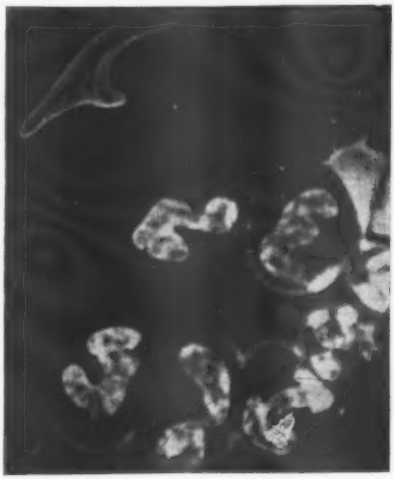

FIGURE XIII. A hooklet with pus cells in the urine. (After Lee Brown.)

FIGURE XIV. A flat plate showing two areas of calcification about a normal pyelogram.

FIGURE XV. The lateral plate shows an anterior hepatic and a posterior renal shadow.

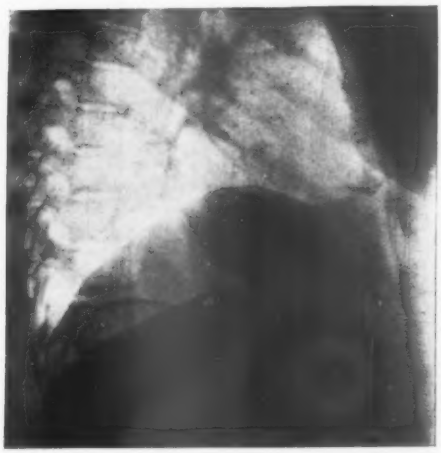

FIGURE XVI. A lateral plate showing the backward position against the spine of a cyst of the posterior surface of the liver. Note the high position relative to the diaphragm.

failure. A mass in the right hypochondrium appeared to be connected with the liver. Autopsy revealed a closed hydatid of the upper pole of the right kidney extending beneath the liver and compressing the bile-duct.

Cysts of the lower pole of the kidney, although markedly displacing the ureter, rarely cause ureteric obstruction. Pseudo-closed cysts are often accompanied by mild intermittent renal pain due to calyx colic. There is often associated a slight hæmaturia. Small calcareous particles may be passed from the exposed adventitia.

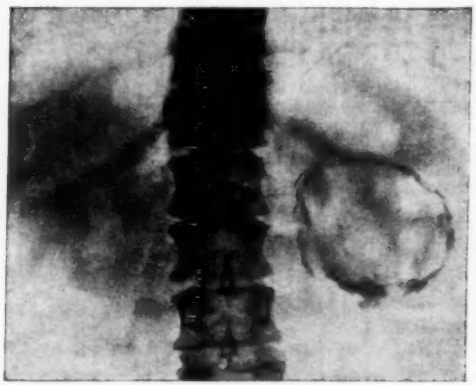

FIGURE XVII. A circular area of calcification in the left renal area.

FIGURE XVIII. Pyelogram showing the wine-glass sign: pseudo-closed cyst. (After Hurley.)

FIGURE XIX. The mottled appearance due to the entry of the opaque medium into a sac containing daughter cysts.

FIGURE XIXA. Open cyst of the lower pole. Deformity of the inferior calyx. Distortion of pelvis and upper calyx from sepsis.

In another personal case the patient had repeated attacks of renal colic from such a cause. The true nature of the disease was unsuspected until urological investigation was undertaken.

In open cysts rupture is often accompanied by severe lumbar pain. This is followed by attacks of ureteral colic from the passage of hydatid débris

down the ureter. Dysuria and even retention of urine may follow the accumulation of membrane in the bladder. Hæmaturia is common. The passage of abnormal urinary constituents is obvious to the patient. Brood capsules and caseous material may be regarded as gravel. Pieces of membrane

FIGURE XX. Open cyst of the upper pole. Blood clot in a hydronephrosis.

FIGURE XXI. Pyelogram of cyst shown in Figure XX. Calcified cyst above a dilated pelvis.

FIGURE XXII. The upper closed cyst pushes the superior calyx aside. The lower cyst opens into the middle calyx.

FIGURE XXIII. Pyelogram of cyst shown in Figure XXII; mottled appearance of the open cyst. (After Begg.)

are described as grape skins. There may be marked shrinkage in the size of the tumour. The rupture may be much less acute, the breach small and the egress of contents negligible. In three cases in this series the first symptom was the passage of a small calculus. The nucleus of the stone in each case was a tiny piece of laminated membrane.

If the cyst becomes closed again, the attacks may not recur for months or years. There may be perfect health in the interim. If the cyst remains open the onset of infection may modify the picture. There may be dull renal pain, with the signs and symptoms of urinary infection. There may be general symptoms from toxic absorption. In open cysts anaphylactic symptoms may occur at any time.

DIAGNOSIS.

Rarely does a renal hydatid cyst come under observation without the presence of a palpable tumour in the lumbar region. The discovery of a smooth rounded mobile tumour in a patient enjoying good health is always significant of hydatid disease. The mass rarely presents posteriorly. A cyst in the upper pole moves upwards under the diaphragm, displacing the kidney downwards. A lower polar cyst usually presents as an abdominal tumour, and displaces the kidney upwards. The cyst is resilient and painless. Its tension is higher than that of a hydronephrosis, but lower than that of a pyonephrosis. The hydatid thrill is rarely felt in renal cysts. The contour may vary. Shrinkage may occur after rupture. Enlargement may follow infection of the cyst.

The passage of hydatid material in the urine is conclusive evidence of infestation in the urinary tract. The various elements of the cyst all possess typical microscopic appearances.

1. The membrane is tough and resistant. It maintains its characteristic laminated structure for long periods, even in the presence of suppuration.

2. The ovoid scolex has a hyaline cuticle. A single row of hooklets, some thirty-six in number, surrounds the head (Figure XII).

3. The hooklets are readily shed. They are small hyaline bodies with one or two root processes. They are commonly found in the urine when there is an open cyst. They are not easily confused with any other abnormal urinary constituent (Figure XIII).

4. Daughter cysts are pearly white, containing an opalescent fluid and scolices. They are rarely passed intact.

Owing to the tendency of a hydatid cyst to calcify, radiography is of particular importance. A flat plate may show a typical rounded area of calcification. It must be remembered that calcified cysts originating in the lower surface of the liver or in the spleen may cast a shadow overlying the renal area. Further examination with lateral plates will show the anterior position of the intraabdominal cysts (Figures XIV and XV).

Cysts of the posterior extraperitoneal liver surface may cause confusion as they will not be projected forwards in a lateral examination. Practically the separation of these from cysts of the upper pole of the kidney is of little consequence. Both are accessible by the extraperitoneal lumbar route (Figure XVI).

Cystoscopy and pyelography are the final stages in diagnosis and localization. Evidence of inflammation and pieces of membrane may be seen in the bladder. In one personal case a piece of membrane was seen to protrude from a ragged dilated ureteric orifice. In another case in which the patient was examined by a colleague, a small daughter cyst was seen to drop from the ureter. The ureteric efflux is often cloudy and the function depressed.

PYELOGRAPHY.

In the pseudo-closed cyst there has been an absorption of exposed adventitia. A potential space has been opened up between the laminated membrane and the remaining adventitia. The opaque medium may fill this

space, producing the typical "wine-glass sign" described by Surrace and Mezzera (see Figure XI).

In the open cysts the "wine-glass sign" is often present (Figures XVII and XVIII). The contrast medium may also enter the cyst cavity. When most of the contents have been expelled the medium may outline the cavity of the sac. Usually it percolates amongst a packed mass of daughter cysts. The cyst presents a typical mottled appearance in the pyelogram (Figure XIX). In old infected cases there may be general distortion from urinary sepsis-clubbing of the calyces and dilatation of the pelvis and ureter (see Figures XIXA, XX and XXI).

Closed cysts present the chief difficulties in diagnosis. They tend to push an obstructing calyx aside. The urinary channel is found as a narrow slit stretched about the convexity of the tumour. If the opaque medium enters the compressed calyx the appearance is of the "spider leg" type similar to that produced by hypernephroma or simple serous cyst (Figures XXII, XXIII and XXIV). If there is no filling of the compressed calyx there is an irregular "broken pipe stem" appearance of the infundibulum. The whole calyx may be amputated from the pyelogram. A closed hydatid does not affect the outline of the renal pelvis until a late stage. The quadrilateral appearance of the pelvis so often seen in malignant cases is absent (Figures XXV and XXVI).

A hydatid cyst of the liver may overlie the renal area and even deform the pelvis from pressure (Figure XXVII). An X-ray examination with the patient in the lateral position will help to differentiate calcified hepatic cysts. A splenic cyst may overlie the left renal area, but, if opaque, is again well forward in a lateral examination. The kidney may be displaced by a splenic tumour.

In one case seen recently the left kidney was low and there was a mild hydronephrosis. Above the upper pole was an indefinite rounded shadow which was disregarded. The kidney was explored and beyond the low position no abnormality was found. A localized bulging of the posterior peritoneum led to the discovery of a hydatid cyst in the spleen (Figure XXVIII).

In the many distortions produced in pyelographs by various types of cyst there are some appearances that are typical of hydatid disease: (*a*) the mottled appearance produced by the entry of an opaque medium into a sac containing daughter cysts; (*b*) any localized deformity of a calyx (and especially the "wine-glass sign") surmounted by a circular area of calcification.

In all pyelograms when the deformity is polar and limited, something short of nephrectomy may be contemplated.

This may be partial nephrectomy or evacuation of the cyst. It is wise to examine all the calyces in the pyelogram. Expansion and partial filling defects within the lumen of a dependent calyx may suggest the presence of a secondary implantation cyst. This may easily remain undetected at a conservative operation (Figure XXIX).

PROGNOSIS.

The progress of the disease is slow, and at any time the parasite may become inactive. However, there is ever present the risk of rupture into the urinary channels with the possibility of anaphylaxis and the probability of infection. If extension of the cyst occurs beyond the kidney, dense adhesions are formed to surrounding structures, which may render subsequent eradication of the parasite difficult or impossible. A conservative attitude may be adopted towards calcified cysts, which the pyelogram shows are not encroaching upon the urinary channels. Particularly is this so in a patient who is a poor operative risk.

TREATMENT.

Extraperitoneal exposure of the kidney by the lumbar route is the method of approach preferred. Removal of the last rib is often necessary in dealing with cysts of the superior pole. The following operative procedures may be briefly reviewed.

Nephrectomy.—The radical operation is nephrectomy. Its performance is not always possible owing to the density of the adhesions. It is the method of choice in the following

FIGURE XXIV. A closed cyst faintly outlined separates the two upper calyces and compresses them.

FIGURE XXV. An irregular shadow of calcification in the left renal area.

FIGURE XXVI. A closed cyst amputating the inferior calyx and encroaching on the pelvis.

FIGURE XXVII. Deformity of the renal pelvis from a cyst of the lower surface of the liver. (After Lee Brown.)

cases: (*a*) when the bulk of the kidney has been destroyed; (*b*) when there is multiple infestation of the kidney; (*c*) in large open and particularly infected cysts. One of the more conservative procedures is suggested when there is any suspicion about the efficiency of the opposite kidney, when nephrectomy is impossible, and in certain types of infestation.

Partial Nephrectomy.—Partial nephrectomy is rarely practicable. Seldom is a cyst small enough to justify the employment of this operation.

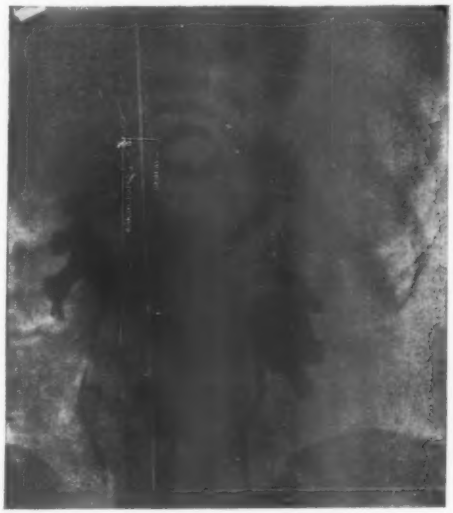

FIGURE XXVIII. Descent of the left kidney due to pressure exerted by a cyst of the spleen.

FIGURE XXIX. Pyelogram of cyst shown in Figure VIII, showing deformity of the superior calyx from a primary cyst and of the inferior calyx from a secondary implantation cyst.

FIGURE XXX. Irregular calcification of a tuberculous pyonephrosis.

FIGURE XXXI. Contrast medium outlining a tuberculous cavity associated with the inferior calyx.

Evacuation and Closure of the Cyst without Drainage.—The wall of the sac is exposed, carefully isolated by packing and punctured with trocar and cannula; the fluid contents are aspirated and measured. The fluid is replaced by a slightly smaller amount of 10% solution of formalin. This is allowed to remain for five minutes in order to devitalize the parasite. Aspiration of the formalin solution is followed by incision of the sac wall. The laminated membrane, if intact, may be removed with forceps in one piece. It is but loosely attached to the adventitia. Usually it lies broken up in the midst of daughter cysts, and a long handled spoon is used to evacuate the contents of the cyst. The cavity, now lined by the adventitia, is swabbed over with formalin, and the incision in its wall is securely closed by suture. This method is particularly suitable in closed cysts, when the contents are clear and therefore relatively aseptic. It is conservative of renal tissue and does not entail mobilization of the kidney. The attendant risks are the overlooking of fragments of membrane or the subsequent filling of the sac with a mixture of blood and urine, which may become infected. The latter risk may be minimized by filling the cavity with saline solution before closure. The cyst wall is sewn to the deeper layers of the wound and a drain placed down to it.

FIGURE XXXII. Carcinoma of the upper pole of the kidney with deformity of the superior calyx.

Evacuation and Drainage.—Evacuation and drainage are reserved for open suppurating cysts when nephrectomy is inadvisable or when there is any doubt about the complete

FIGURE XXXIII. Serous cyst of the kidney spreading the two upper calyces.

FIGURE XXXIV. A cyst opening into the right lateral wall of the bladder. The opaque medium has the mottled appearance showing the presence of daughter cysts.

removal of the contents of a sac. Drainage involves the risk of secondary hæmorrhage, and may entail the persistence of an infective or a urinary fistula.

SUMMARY.

The life cycle of the hydatid parasite and the mode of human infestation have been described. Involvement of the kidney is uncommon.

The evolution of the cystic stage in renal tissue is subject to modification owing to the presence of the urinary channels.

Systemic effects of hydatid disease form the basis of the reliable laboratory procedures—the Casoni and complement fixation tests. When the cysts are closed, the symptoms are often obscure and the diagnosis is difficult. Open cysts are often associated with suggestive symptoms and reliable clinical findings.

The special diagnostic methods of radiography and pyelography provide useful information regarding the nature and localization of the cysts.

Methods of operative procedure have been briefly reviewed.

The material from which this paper was drawn is contained in the records of the Hydatid Registry of the Royal Australasian College of Surgeons and a personal series of seven cases.

ACKNOWLEDGEMENTS.

My thanks are due to Dr. Edgar Thomson, pathologist at the Royal Prince Alfred Hospital, Sydney; to Dr. Neil Guthrie, radiologist at the Christchurch Hospital, and to Mr. Victor Hurley for permission to use one of his cases. I am under a debt of gratitude to Sir Louis Barnett for his unfailing help.

THE SIGNIFICANCE OF JAUNDICE, INFECTION AND ACIDOSIS IN CALCULOUS OBSTRUCTION OF THE COMMON BILE DUCT.[1]

By W. A. Hailes,
Melbourne.

In urinary obstruction due to prostatic enlargement, treatment does not commence and end with the removal of the prostate; in calculous obstruction of the common bile duct, treatment does not commence and end with the removal of the stone. In the former case, obstruction leads to chronic pyelonephritis, in the latter to biliary cirrhosis with all its accompaniments—namely, upset in carbohydrate metabolism, decreased sugar tolerance, impaired biliary function culminating in the appearance of so-called white bile, deficient prothrombin formation with a deficient coagulation of the blood, impaired fat metabolism with deficient fatty acids in the circulation.

These are the accompaniments of "the liver of surgical delay". How are cases of this condition to be managed? What is the significance of outstanding clinical entities—for example, jaundice, infection, acidosis?

Jaundice.

The old teaching and conception that in cases of calculous obstruction of the common bile duct jaundice and cholangitis should be allowed to subside and operation undertaken in an interval of freedom, has in the past few years been questioned by many surgeons. With a more detailed knowledge of the changes that occur in the liver as the result of calculous obstruction and of the manner in which they jeopardize life, it is necessary not only to review results and statistical tables in an endeavour to determine whether earlier intervention will lower the mortality, but especially to analyse each failure closely to determine whether other methods would have offered more hope of success. Only by this means can we hope to improve our results.

If all risks are accepted, the mortality in cholecystitis and cholelithiasis limited to the gall-bladder is between 2% and 5%. When a stone or stones are present in the common bile duct unaccompanied by jaundice, the mortality is very little higher.

Jaundice is the only reliable symptom of biliary obstruction. It must be remembered that hepatic function may have been impaired by an incomplete obstruction for months or years before the onset of clinical jaundice. The stage of cirrhosis of the liver may have been reached when jaundice appears. In the days when Van den Bergh tests were more commonly carried out than they probably are today, I saw several patients who had yielded positive reactions but were not clinically jaundiced; and I have vivid recollections of one of my medical colleagues standing over me till I found the stone which I had at first said was not present—and this in spite of the fact that I had opened the common bile duct. That stone was in the ampulla, was of some size and the obstruction to the flow of bile must have been present for months. The presence of a slight degree of jaundice raises the mortality to 5%. This figure and that which follows will obviously depend on the interpretation of the terms slight and severe, but with severe jaundice and dark

[1] Accepted for publication on April 27, 1939.

bile in the common bile duct the mortality rises to 15% in some clinics; with white mucoid fluid in the ducts it rises to as high as 30%.

As it is impossible in most of these cases to separate obstruction and infection, it does not seem quite correct to discuss the total mortality under the heading of jaundice. It has been emphasized that jaundice is the only indication of biliary obstruction. It must now be stated that 20% to 25% of patients with stones in the common bile duct, even including many whose stones are large, multiple and of long duration, have never been clinically jaundiced. In an analysis of my own cases or of published statistics, I have found it exceedingly difficult in some of the cases to estimate which was the outstanding factor, obstruction or infection.

Unless the obstruction has been followed by the stage of gross suppression of hepatic function, severe infection is the more significant.

If general health admits of operative interference, three factors which are of outstanding importance in most cases must be considered. These are the presence or absence of (*a*) jaundice, (*b*) infection, (*c*) acidosis.

How does jaundice influence mortality? Jaundice is a clear indication of biliary obstruction which may, in a mild degree, have been present for months or years. The present state is an exacerbation possibly amounting to complete obstruction.

In the presence of jaundice there are (*a*) a tendency to hæmorrhage, (*b*) a risk of impairment of liver function, (*c*) a problematic factor—the degree of improvement of hepatic function while the stone remains.

While the tendency to hæmorrhage in jaundiced patients is always a disturbing factor and a destroyer of rest, I am certain that the risk of operation is not very great in calculous cases if a minimum of interference is carried out. The risk of hæmorrhage in these cases is not so great as in obstruction from carcinoma of the head of the pancreas or other malignant disease.

In regard to impaired liver function, if the old teaching is followed and we delay until the jaundice disappears, what is our margin of error? Unfortunately, these changes do not run on a time schedule. The back-working of bile may have been present for years, months at least, before the onset of complete obstruction was indicated by jaundice and was followed quite quickly in some cases by suppression of bile and the appearance of a mucoid fluid in the biliary tract.

My last two cases of calculous obstruction of the common bile duct associated with jaundice and white mucoid fluid in the biliary tract are instructive because of the short duration of jaundice in each case.

Mrs. T., aged seventy years, had had recurrent attacks of upper abdominal pain for some years and the last one, which commenced two and a half weeks previously, was associated with jaundice. The jaundice had not varied in intensity; there had been no shivers or sweats.

On examination, her temperature was 98° F., and her pulse rate was 84 in the minute. Her tongue was clean. A moderate degree of jaundice was present. Her systolic blood pressure was 170 and her diastolic pressure was 105 millimetres of mercury. The liver was enlarged to four fingers' breadth below the costal margin. The gall-bladder was not distinguishable. The urine was dark; it contained a trace of albumin, but no sugar, acetone or diacetic acid. The fæces were clay coloured.

Two days were taken in preparation and then operation was performed. Cholecystostomy and choledochostomy were carried out for stones in the gall-bladder and in the common bile duct. White mucoid fluid was present in the common bile duct, and it was days before the bile was reasonably coloured.

The patient was discharged from hospital in one month, and should have been operated on at least fourteen days earlier.

Mrs. H., aged forty-five years, was admitted to hospital on November 26, 1938. She had had a normal confinement three months previously. Three weeks prior to her admission to hospital she developed severe colicky abdominal pain, biliary in character.

Almost every day since the onset she had had similar attacks of pain, though this was not nearly so severe as latterly. There had been no vomiting, shivers or sweats. Five days prior to her admission, on November 22, 1938, jaundice was evident and the urine was dark.

On examination the liver was palpable two fingers' breadth below the costal margin; the gall-bladder was not palpable. On X-ray examination an oval shadow half an inch in diameter was seen opposite the level of the disk between the first and second lumbar vertebræ; it was regarded as a gall-stone.

Operation was performed on December 6, 1938, fourteen days after the onset of jaundice. A stone was removed from the lower end of the common bile duct and the gall-bladder was drained. The ducts contained a white mucoid fluid and no bile coloured fluid was discharged during the operation. Two days later the fluid drained from the common bile duct was fairly deep in colour, though not quite normal. On January 5, 1939, the patient was discharged to a convalescent hospital.

In these cases there was complete suppression of liver function three weeks and two weeks respectively after the onset of clinical jaundice. The free oral administration of bile in salol coated tablets as indicated by R. D. Wright in THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, April, 1933, is essential in such cases.

Some surgeons have recently gone so far as to insist that the onset of jaundice is an indication for operation and not an indication for delay. This is largely because of the impairment of hepatic function. As long as immediate operation is not advocated, I am in agreement.

If, as has been stated, the tendency to hæmorrhage is not insurmountable, the chief difficulty is some risk of hæmorrhage from adhesions and some risk of hæmorrhage and of difficulty in dissecting the common bile duct because of the inflammation in its wall and surroundings. This is not often marked, but may be disconcerting. My view is distinctly that if in a few days the jaundice is not becoming less, it is an indication for operation. If the jaundice is decreasing it is well to wait till the pigmentation has disappeared and the fæces and urine are normal.

The influence of infection will be considered later.

How do you control the risk of hæmorrhage? By doing a minimum. These patients are not good operative risks; a live patient is an asset, and there is always another day. The bleeding from the abdominal wall can usually be controlled and the same applies to the adhesion and the wall of the common bile duct; vessels here can be ligated or the wall of the duct can be sutured. It is the liver bed which bleeds. If in any doubt, do a minimum—cholecystostomy and choledochostomy, or choledochostomy, dilate the duct and drain it.

INFECTION.

How does infection influence mortality? The old teaching of delay seems to me to be fraught with much greater risk in the presence of obstruction and infection than with obstruction only. Is it reasonable to expect that a really acute cholangitis will certainly subside till the obstruction is removed? We know that it often will, but the following case in which no operation was performed and death occurred, shows what may happen.

Mrs. D., aged forty-seven years, gave a history that two days prior to my seeing her there had been an acute onset of severe upper right-sided abdominal pain. Twenty-four hours after the onset, and one day before I saw her, the patient had had a shiver and sweat; these had been severe and followed by faint jaundice. Her temperature was 103° F. and the pulse rate was 120 in the minute. Great tenderness and rigidity were present over the upper part of the abdomen on the right side. No mass could be felt. Her tongue was coated and moist. Her systolic blood pressure was 140 and her diastolic pressure 80 millimetres of mercury. The urine contained no albumin, no sugar, no acetone or diacetic acid. Bile was present in the urine. The heart and lungs were clear. The patient had had one previous severe attack two years before. A diagnosis of acute cholecystitis with possibly cholangitis was made. Operation was advised.

As the temperature subsided a little next day and the general condition improved, operation was declined. Twenty-four to thirty-six hours later the condition rapidly deteriorated with, I am sure, the development of a suppurative cholangitis with hyperpyrexia and extreme jaundice. The patient was never again operable because of the general condition.

This patient's common bile duct should have been opened and drained on the day I first saw her or on the following day.

Is there any risk that the diagnosis is incorrect and that the true condition is a suppurative cholecystitis with involvement of the common bile duct? I have seen this several times, and the condition present at operation was one that should have been dealt with days before.

Mrs. B., aged sixty-eight years, on December 25, 1935, became ill with extremely severe right-sided upper abdominal pain radiating to the back and associated with vomiting and a rigor. Her temperature was 104° F., and her pulse rate 120 in the minute. On examination twelve hours later she was icteroid, her temperature was still 104° F.; her pulse rate was 112. She had had gall-bladder attacks for years.

On December 26, 1935, I saw her for the first time. There had been another rigor lasting twenty minutes; the patient was now definitely jaundiced and was delirious. Her temperature was 103° F., and her pulse rate 120. The tongue was dry and brown. The urine contained a trace of albumin, but no sugar; acetone was present. Glucose and saline solution were administered *per rectum* and intravenously.

On December 27, 1935, the patient had another rigor just prior to operation, which revealed a quantity of free fluid in the peritoneal cavity. A large gall-bladder full of stones and offensive bile and a large common bile duct containing stones, mud and infected bile were found.

This reveals the impossibility of an accurate diagnosis. My diagnosis was infective cholangitis from calculi in the common bile duct. I did not anticipate that I should find an empyema of the gall-bladder and free fluid in the peritoneal cavity as well. The patient made a surprisingly good recovery.

Delay endangers life in these cases and one can only plead that mistakes in diagnosis will always occur. Is there an analogy anywhere else in the body? Quite frequently a stone blocks a ureter and some days or weeks are allowed to pass before operative intervention is carried out in the hope that all will subside and the stone pass. But let a stone block the only functioning ureter; everyone then knows that relief is essential within the first few days. Here is an analogous condition: the duct of a single essential gland blocked. The time factor may be more important and secretion is at a higher pressure in the renal case, but the conditions are analogous in other respects.

What is gained and what might be lost by operating in these stages? And, again, what do you do?

The gain is an adequate drainage of the larger biliary channels and reduction of infection with diminution of toxæmia, and we hope, if the cholangitis has not been too severe, that restoration of hepatic function will occur. I can see very little that is lost, except possibly that there is some increased risk of post-operative pulmonary complications.

The hypothetical possibility that the infection will spread is not a very real one; these patients after operation do not die of a general peritonitis. Some degree of infection will be present whenever the operation is performed.

The risk of an exacerbation of cholangitis due to operation is small indeed compared with the relief obtained from adequate drainage. It must be admitted that the risks of post-operative pulmonary complications are greater with acute lesions in the right upper quadrant of the abdomen than with chronic lesions. Because of the inflammation the diaphragmatic movement on that side is restricted.

What is done? Again a minimum. These patients are bad operative risks. Cholecystostomy is performed, and in these cases, if the infection is severe, choledochostomy—not just choledochotomy; dilatation of the ampulla is

carried out as is done sometimes when the infection is not severe. For T-shaped tubes and permanent or prolonged drainage I have little regard once the acute infection has subsided. The sooner the bile is once more discharged into the bowel and the fistula closed, the better.

Some surgeons, although advocating immediate operation in the presence of biliary obstruction, counsel delay in the presence of infection. Infection in these cases is obviously associated with obstruction, the obstruction being either in the common bile duct or the smaller ducts or both. Surely obstruction with infection is a much greater danger to the patient than obstruction alone. Relief can be hoped for only by natural subsidence of the inflammation, which is problematical, or by surgical aid, removal of the stone and drainage. If other factors permit, this must be done.

ACIDOSIS.

In regard to acidosis, it is not enough to test the urine of patients suffering from obstruction of the common bile duct for albumin and sugar. Acidosis is a real danger and in some cases is due to much more than a deficiency of carbohydrate intake because of vomiting. In severe cases glucose given by mouth will be excreted in the urine unless insulin is given. Patients who require insulin prior to operation may on discharge from hospital pass sugar-free urine while on a full diet, indicating that the acute upset of carbohydrate metabolism is a complication of the disease.

Mrs. M.K., aged seventy-one years, seen on October 24, 1935, gave a history of attacks of upper abdominal pain for forty years. The pain occurred on the right side, radiated through to the back and made her roll about. Five days before she was seen she had a very severe attack of pain associated with a shiver and sweat. Jaundice occurred in this attack and had been present with some of the other attacks.

On examination her temperature was 101° F., and her pulse rate was 120 in the minute. Her systolic blood pressure was 146 and her diastolic pressure 68 millimetres of mercury. Her tongue was dry and coated. Her breath was acidotic. She was very drowsy. Tenderness was present all over the abdomen, but it was worse in the right upper quadrant. Examination of urine revealed a trace of albumin, but no sugar; the urine was loaded with diacetic acid and acetone. She was given glucose by mouth, and in twelve hours the urine was loaded with sugar, acetone and diacetic acid. The blood sugar content at 8.45 a.m. on October 26, 1935, was recorded as 0·45%.

Dr. S. O. Cowen took the patient in charge and as the inflammatory state subsided time was not a factor of importance. It required eighty-four units of insulin daily (thirty, twenty-four, thirty) to keep the patient's urine sugar-free during the pre-operative period.

Operation was performed on November 12, 1935, and comprised cholecystostomy and choledochotomy. The patient was discharged on December 8, 1935, with sugar-free urine on a full diet without insulin.

In otherwise suitable cases the one positive contraindication to operation is acidosis; this is not infrequently present and should always be sought.

Fortunately today acidosis can be readily controlled. Mild cases respond readily to medical treatment with adequate dietary, fluids and insulin. More serious cases, in which the acidosis is extreme and the cholangitis severe and need of operative intervention is more urgent, can be controlled by continuous intravenous administration of glucose and injections of insulin.

It seems imperative that we should emphasize the view that the teaching must be altered. No longer must we consider jaundice in calculous disease a contraindication to operation. Rather, if it persists for more than a few days, must it be considered an indication for operation. Jaundice only emphasizes the fact that a pathological state that has in many cases been in existence for a long time, has reached a climax.

Cholangitis must be considered an indication for operation—not an indication for delay; there is ample time for dehydration and acidosis to be controlled if modern methods are adopted.

POST-OPERATIVE PULMONARY COMPLICATIONS.

In the management of acute lesions of the biliary tract I have learned to fear the consequences of post-operative pulmonary complications just as much if not more than the technical difficulties of the operation and the post-operative management of the abdominal condition.

Pulmonary complications are the reason why I cannot bring my mortality rate for acute cholecystitis down to between 2% and 3%.

My last two fatalities with acute cholecystitis were due to post-operative pulmonary complications. In these cases I had the help of a skilled physician; the anæsthetic in each instance was ethylene and oxygen given by an expert anæsthetist. Pulmonary complications have not been such a feature with cases of calculous obstruction of the common bile duct, though many of the patients have been elderly and stout. Judd has said that pulmonary complications are three times as numerous as all other complications in gall-bladder surgery and correspondingly severe.

In the hands of a competent surgeon and expert anæsthetist I feel sure that in cases of calculous obstruction of the common bile duct, jaundice and infection are indications for operative intervention once dehydration and acidosis are controlled.

What causes me to sound a note of warning is only the fear that a prolonged operation under an indifferently administered anæsthetic may have such dire consequences that more may be lost than gained. It is imperative to emphasize the standards required; if these conditions are available, operative intervention is indicated.

THE ADVISABILITY OF MORE GENERAL EXPLORATION OF THE COMMON DUCT.[1]

By Balcombe Quick,
Melbourne.

The responsibility for the ultimate failure of an apparently successful operation for cholelithiasis may, on occasions that are by no means rare, be ascribed to the overlooking of a stone in the common duct. I have not been immune from such failures and I venture to doubt if I am alone in this respect.

It will be agreed that it may be a very difficult matter to arrive at a decision as to the likelihood of such an obstruction being present, either after the fullest consideration of symptoms, physical findings and radiographic examination, or even after the abdomen has been opened and the gall-bladder exposed.

A recent survey of my own hospital cases over the last ten years, undertaken for me by the Surgical Registrar, Dr. Burnside, whose help I gratefully acknowledge, has served to crystallize certain impressions that I have been forming for a number of years. The series is not a large one, for reasons into which it is not necessary to enter, but is sufficient, I believe, to allow certain conclusions to be drawn from it.

In this series of 218 cases a single stone or multiple stones inhabited the common or hepatic duct in 37, the common duct in the great majority, that is in 17%, so that it may be said that in one case in every six a choledochotomy is an essential part of the operation.

The problem, then, is to recognize those cases in which choledochotomy is demanded. The complete picture of Charcot's intermittent hepatic fever is, in these enlightened days, comparatively seldom met. A history of past or present jaundice, although obtained more frequently from those harbouring a duct stone than from those without, is not an essential feature. For in seven of the 37 cases with duct stone any history of jaundice was entirely lacking. The jaundice that had been noted in 30 cases, representing 81%, is not strikingly dissimilar in its incidence from that accompanying simple cholelithiasis, nearly 70% of 181 cases. No reliance, therefore, is to be placed upon a history of freedom from jaundice.

From quite a number of those patients accepted as giving a positive history nothing more definite than darkening of the urine, lightening of the stools or a muddy complexion had been elicited.

Without going more fully into the matter and wearying you with figures I would simply say that we have found neither the type of pain nor its radiation nor anything else in the clinical picture of any particular value in the detection of the common duct stone case.

At operation it may be equally difficult to be sure of the duct, at any rate of the important lower end, by any methods of palpation. Nor is the fact that the gall-bladder, whether it be large and distended or small and sclerotic, contains no sign of calculus in any way conclusive that the common duct is equally empty and patent. In at least six cases in the series such a state of affairs existed. The dilatation or otherwise of the common duct observed to be present is, to my mind, not very conclusive. Whilst gross dilatation is undoubtedly of the very greatest significance, a minor degree

[1] Accepted for publication on May 9, 1939.

speaks neither for nor against stone, particularly if the gall-bladder be shrunken and fibrous.

For these reasons I have come to explore the duct by choledochotomy more and more. I have come to ask myself, not what are the indications for exploration, but what are the indications for refraining from it. I seek to justify this attitude, firstly by drawing attention, as I have done, to the difficulties of certainty in the diagnosis, and then by a statement of results. Such diffidence as may be felt about a rather general exploration of the common duct, is largely due to published figures of the mortality rate attaching to the operation of choledochotomy. The mortality is that of the pathological condition for which the operation was undertaken rather than of the procedure itself.

Of the 218 cases in question, the duct was opened and explored in 148; the operation was confined to the gall bladder in 65, and in five cases the history is silent as regards the duct.

The mortality of the whole series was 4·6%. Three deaths occurred amongst the 70 cases in which the record shows that the operation was confined to the gall-bladder, the mortality here also being 4·6%. In 148 cases, including those with the more advanced pathological changes in the liver and elsewhere, in which the duct was explored, the mortality to the nearest decimal point was 4·7%.

I shall not go further into comparative figures beyond saying that a "completely uneventful convalescence" resulted in 45·3% and 49·2% respectively.

Of the deaths in this series, three were due to coronary disease, one each to intestinal obstruction, associated carcinoma of the stomach and liver abscess, pulmonary abscess, undiagnosed pyonephrosis and uræmia; two deaths were due to cardiac failure, one on the sixth day and the other after twelve hours; and one was due to an undiagnosed subphrenic abscess.

A very critical survey of these ten deaths with reference to what part a fruitless exploration of a common duct and the resulting prolongation of the operation may have played in a fatal result, directs attention to one case and one case only—that in which death occurred twelve hours later.

This patient had undergone cholecystotomy for solitary stone some years before in another hospital. With a continuance of attacks of pain and a large gall-bladder containing no stone, it seems in retrospect that no course was open to me but to undertake an exploratory choledochotomy. The fatal result was due to an exceptionally difficult and bloody cholecystectomy in a patient whose condition gave rise to anxiety from an early stage of the operation and whose condition failed to improve following blood transfusion.

Even if this death be laid at the door of a fruitless choledochotomy, I would set against it those several cases in which a more or less unexpected obstruction was detected and removed—unexpected because the gall-bladder was found to contain no stones and the classical symptoms of common duct obstruction were lacking.

In regard to exploration itself—its technique—I continue to look upon Desjardin's forceps as my best asset, not only for removal of the stone, but for ascertaining its presence. The small tell-tale adherent fragments may be of the greatest value. In the rare instances in which this instrument fails in the attempted removal of a stone impacted at the ampulla, a transduodenal approach should be resorted to without the slightest hesitation. On two occasions in this series this became necessary, and the results were satisfactory.

CONGENITAL CYSTIC DILATATION OF THE COMMON BILE DUCT.[1]

By Hugh R. G. Poate,
Sydney.

With the case now reported a review of congenital cystic dilatation of the common bile duct is presented and in all 105 cases are briefly detailed.

This article is based on that published by Zinniger and Cash in the *Archives of Surgery*, Volume xxiv, 1932, with the addition of cases reported since 1930.

There has been a good deal of variation in the name by which this condition is to be known and after a careful review of the literature, and in particular of the ætiological consideration, it appears to me that Zinniger and Cash were correct in their arguments in favour of the name used in the title of this article.

It seems essential to recognize that the condition is congenital in origin and that symptoms or signs first occur as a rule in childhood or early adult life; in fact approximately 90% of the undoubted cases were found in persons under thirty years of age when diagnosis was established and most of the adults had symptoms going back to their childhood. The condition appears to be more common in females than in males, but there is no apparent reason why this should be regarded with any special significance.

The lesion present is not a diverticulum of the common bile duct as it involves the whole length of the duct and its whole circumference; nor is it a cyst, but may be termed "cystic" since it is relatively thin-walled and is full of fluid bile.

When one considers the normal anatomy of the common bile duct it must be apparent that when gross dilatation occurs the pylorus and duodenum must be lifted forwards and medially, while the right flexure of the colon is pushed downwards. The main enlargement develops in the right side of the abdomen and clinically produces a cystic swelling below the right costal margin and in the right flank.

The principal diagnostic triad is: (*a*) the presence of a cystic swelling in the right side of the abdomen, (*b*) jaundice and (*c*) pain, but they are not all always present. In acute cases jaundice is apparently produced by kinking or pressure upon the intraduodenal portion of the common bile duct, while pain is most likely due to the increased tension possibly augmented by infection.

It is of interest to note that in 27 out of 34 post-mortem examinations attention was drawn to the patency of the intraduodenal portion of the common bile duct, and incidentally it may be noted that the dilatation does not affect this part of the duct as a rule. In old-standing cases, such as that reported by McWhorter, there may be secondary dilatation of the hepatic duct, the intrahepatic bile channels and even of the pancreatic duct.

In many cases infection had occurred; it was much the same as that met with in empyema of the gall-bladder with cystic duct obstruction, and is very likely secondary to stagnation of the bile. In Saint's case there was a pure culture of *Bacillus typhosus*.

[1] Accepted for publication on December 19, 1940.

In some cases the gall-bladder and cystic duct have been reported as being dilated, but this is by no means a feature of the condition and seldom occurs in the acute cases in young patients.

The cirrhosis of the liver and splenic enlargement simulating the condition found in Banti's disease seem to be secondary to the chronic obstruction to the bile flow, as well as to pressure on the portal vein by the dilatation which varies so greatly. Attention is drawn to two unusual cases. The first is that reported by Blocker (Case 84A, Table II); in this case there had been a traumatic rupture of the lesion in a boy of fourteen years who fell from a swing across the back of a park seat and had immediate signs of a ruptured viscus. Although operated on within a few hours, he died next day. The other case was that of Swartley and Weeder (Case 93, Table II), who in a boy of four years found that there was a double common bile duct with congenital cystic dilatation confined to the one on the right side, which was excised deliberately. Some four years later this boy was subjected to a splenectomy and it was noticed that the right lobe of the liver was atrophic, whereas the left lobe had hypertrophied. It is interesting to note that he had developed a splenomegaly and was well two years after splenectomy.

TREATMENT.

As will be seen from Table I, the only treatment that is likely to save the patient's life is (*a*) a primary choledochoduodenostomy as first advocated by Bakes in 1907, but in which it is essential to provide an adequate stoma, or (*b*) in a few cases, drainage for a short period followed by the correct

TABLE I.

Types of Treatment in Cases Reported in the Literature.

Treatment.	Number.	Died.	Recovered.
No treatment	11	11	0
Aspiration	4	4	0
Drainage only	30	28[2]	2[3]
Drainage followed by secondary anastomosis	17	5	12
Primary anastomosis of cyst to intestine	27	7	20
Excision of cyst; primary anastomosis of hepatic duct to intestine	4	2	2
Excision of cyst with drainage; secondary anastomosis to intestine	1	0	1
Excision or attempted excision of cyst, with or without drainage	8	7	1[1]
Nature of operation not known	3	2[4]	—
Total	105	66	38

[1] This was a case of double duct with right side only involved. This was excised deliberately (Case 93).

[2] Three patients died after from one to three months of hæmorrhage from fistula. One was a case of rupture due to trauma (Case 84A).

[3] One patient (No. 14) died after three years, of pulmonary tuberculosis; one was well for four years (No. 42).

[4] The outcome of one case was not known.

Of the twenty-three new cases now added in three the patients were treated medically, one by aspiration and four by drainage only; all died. Of the nine cases in which a primary anastomosis was performed seven patients recovered, as did the three subjected to primary drainage and secondary anastomosis.

anastomosis. This latter procedure should be followed only if the patient is in a greatly debilitated state or when it is obvious that infection has occurred. During the period of drainage it is advisable to administer bile salts in adequate quantity according to the age of the patient and also vitamin *K*, otherwise hæmorrhage may occur.

To quote Zinniger and Cash:

The most encouraging finding brought out by the cases recorded in the literature is that, on the whole, the treatment adopted in the more recent cases has been better than used in the earlier ones. The diagnosis should not be difficult if one bears in

mind that congenital cystic dilatation of the common bile duct may exist, for the clinical symptoms and the physical findings are fairly characteristic. It is to be hoped, therefore, that in the future a correct pre-operative diagnosis may be made more frequently, and that there will be still further improvement in the treatment and results.

CASE HISTORY.

The case to be reported is that of E.F., female, aged four years and nine months. There was nothing significant in her history until six months ago, except for two attacks of skin irritation in the previous six months along with the passage of clay coloured stools.

The present illness commenced in January, 1940, with abdominal pain, vomiting, high temperature and loose motions lasting several days. Three weeks later she had further vomiting and was seen to be jaundiced. The doctor who saw her pointed out that the abdomen was swollen and thought that she might have had an enlarged liver or a hydatid cyst. She was placed in hospital for observation, and a distinct swelling was noted in the upper part of the abdomen on the right side. The jaundice persisted with clay coloured motions. Different tests for hydatid disease gave negative results. X-ray examination showed a cyst, thought to be connected with pancreas or liver.

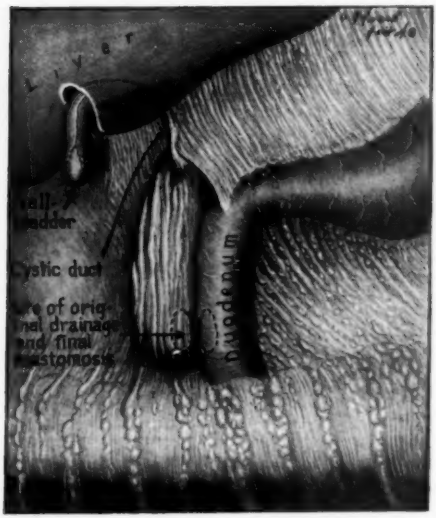

FIGURE I. Showing schematic representation of condition found at the second operation.

At laparotomy early in February a large cyst was found containing some 1,500 cubic centimetres of bile. As diagnosis was uncertain, the cyst was drained. There was a copious discharge of bile, and on the second and fourth days free hæmorrhage occurred necessitating blood transfusions. She was given bile salts by mouth and gradually recovered, leaving hospital at the end of March. In June she was referred to Sir Robert Wade, who asked me to see her. She was discharging some twenty ounces of bile daily and was still taking her bile salts. She was given two weeks in hospital on vitamin *K* with bile salts, and an effort was made to mitigate the infective cholangitis present.

Laparotomy was performed on July 22, 1940 (see Figure I), the cyst wall being detached from the abdominal wall. It was collapsed with puckered antero-lateral walls. The duodenum was attached to its antero-medial aspect and was very mobile. The gall-bladder was small and empty, with the cystic duct joining the upper end of the swelling, which went up to the portal fissure and obviously included the common hepatic duct. The lower limit extended to the middle of the descending part of the duodenum. No attempt was made to find the lower end of the duct, but the edges of the fistula were excised, an anastomosis was made with the duodenum and the abdomen was closed after the insertion of a tube along the track of the sinus through the abdominal wall. Apart from some infection in the abdominal wall, she made a good recovery and had normal stools from the first day after operation.

REVIEW OF LITERATURE.

The most complete reviews of the subject have been presented by Waller (1917),[17] McWhorter (1924),[18], Sénèque and Tailhefer (1929),[16] Zinniger and Cash (1932),[86] Gross (1933),[85] Yotuyanagi (1936),[84] and Walton (1939).[83]

These writers have noted certain discrepancies in cases recorded, and it is questionable whether some of them can be regarded as undoubted congenital cystic dilatation of the common bile duct. Access to some of the literature is difficult to obtain, and some reports are vague as to detail.

TABLE II.[1]

Summary of Twenty-three Cases Reported or Included since 1930.

Note.—The year given is the date cases were seen, but not necessarily the year in which they were reported.

Number and Author.	Year.	Sex.	Age.	Tumour.	Jaundice.	Pain.	Diagnosis.	Treatment.	Result.
84. Berkley, H. K.[73]	1932	F.	10	+	+	+	Biliary obstruction.	Cholecyst-duodenostomy.	Recovery. Well four years later.
84A. Blocker, T. G., and others[74] .. (Numbered as 84A in order to keep consecutive numbers by omission of Case 53 by Reel and Burrell. See context.)	1935	M.	14	–	–	–	Ruptured viscus.	Drainage.	Died next day. This is the only recorded case of traumatic rupture of a choledochus cyst. (See context.)
85. Burman, G. E. L.[75] .. .. (Omitted from previous records, but Walton considers this an authentic case, and I agree.)	1911	F.	17	+	++ Also two years before.	–	Obstructive jaundice.	Cholecystostomy (1½ pints of bile removed).	Died one week later.
86. Frizelle, E. R.[76]	1932	F.	29	?	Once as a child.	+	Acute cholecystitis.	(1) Drained six weeks. (2) Cholecyst-duodenostomy six weeks. (3) Choledochoduodenostomy.	(1) Sinus persisted in draining bile. (2) Sinus persisted in draining bile. (3) Recovery.
87. Fowler, R. S.[3] .. .. .. (Not included by Zinniger and Cash as thought to be secondary to the pancreatitis, but Gross, Judd and others regard it as a true case, and I agree.)	1915	M.	22	–	?	+	Acute cholecystitis.	Cholecystectomy. Drainage of cyst.	Death in three weeks from asthenia. All bile drained away.
88. Gross, R. E.[23]	1929	F.	4½	+	+	+		Choledochoduodenostomy.	Recovery. Well four years later.
89. Gross, R. E.	1931	F.	3	+	+	–	Neoplasm.	Excision and drainage.	Died seven hours later.
90. Hamilton, Ian[77]	1938	F.	20	+	–	++	Acute cholecystitis.	Cholecyst-gastrostomy.	Recovery.
91. O'Gorman, F. M., and others.[78]	1930	F.	8	+	+	–	Hypernephroma.	(1) Excision and drainage. (2) Cholecyst-duodenostomy. Drained.	Died.
92. Saint, J. H.[80]	1931	F.	43	–	–	++	Perforated ulcer.	Drained.	Died two weeks later. Bile infected with *Bacillus typhosus*.
93. Swartley, W. B., and Weeder, S. D.[81]	1932	M.	4	–	–	+	? Intussusception.	Excision.	Recovery. Drained bile for two months. Well two years later. This is the only recorded case with a double common duct. Splenectomy 1936. Well 1938.
94. Upcott, H.[82]	1926	F.	30	+	–	+	?	(1) Drainage six weeks. (2) Choledochoduodenostomy. (3) Two years later abscess in liver drained. (4) Three months later anastomosis was reconstructed.	Recovery. Died six months later with suppurative cholangitis.

[1] Table II and the numbers of the cases recorded are to be regarded as a continuation of the similar table by Zinniger and Cash.[56]

TABLE II.—*Continued.*

Summary of Twenty-three Cases Reported or Included since 1930.—Continued.

Note.—The year given is the date cases were seen, but not necessarily the year in which they were reported.

Number and Author.	Year.	Sex.	Age.	Tumour.	Jaundice.	Pain.	Diagnosis.	Treatment.	Result.
95. Walton, Sir Jas.[83]	1926	F.	16	+	–	–	Splenic anæmia.	Choledochoduodenostomy and splenectomy.	Died twenty-four hours later.
96. Walton, Sir Jas.[44]	1932	F.	34	+	+	+	Obstructive jaundice.	Choledochoduodenostomy.	Recovery. Well six years later.
97. Walton, Sir Jas.[44]	1938	F.	25	+	–	+ +	Choledochus cyst.	Choledochoduodenostomy.	Recovery. Well one year later.
98. Walton, Sir Jas.[44]	1939	M.	39	+	+	+ +	Choledochus cyst.	Choledochoduodenostomy.	Recovery.
99. Walton, Sir Jas.[44]	1939	F.	43	+	+	+ +	Chronic cholecystitis.	Choledochoduodenostomy.	Recovery.
100. Walton, Sir Jas.[44]	1911	F.	1 mth.	+	+	?	?	———	Post-mortem finding.
101. Weber, F. Parkes[87]	1930	F.	12	+	+	+	Cirrhosis of liver.	Medical only.	Died early in 1934. Post-mortem examination revealed rupture of a huge choledochus cyst.
102. Yotuyanagi[94]	1933	M.	2½	+	–	?	Choledochus cyst.	Aspiration (capacity 1,600 cubic centimetres of bile).	Died in three weeks with hæmatemesis and cholangitis.
103. Yotuyanagi[94]	1929	F.	22½	+	+	+	Choledochus cyst.	Cholecystectomy. Choledochoduodenostomy.	Died twenty-four hours later, 5,500 cubic centimetres of bile aspirated.
104. Yotuyanagi[94]	1935	F.	12	+	+	+	? Tumour of liver.	Nil. (Capacity, 2,250 cubic centimetres of bile.)	Died in two months in hospital. Post-mortem diagnosis.
105. Poate and Wade	1940	F.	5	+	+	+	Pancreatic cyst.	(1) Drainage. (2) Anastomosis to duodenum.	Recovery.

Zinniger and Cash have submitted the most exhaustive criticism, and in general their conclusions are sound. However, the case recorded by Reel and Burrell[38] and accepted by other writers to date should be excluded for many reasons (see later), and cases recorded by Fowler[1] and Burman[75] seem to be definite and are now included.

It seems reasonable to agree with Zinniger and Cash in excluding cases of cylindrical or cystic enlargements, such as may occur after partial or complete occlusion of the common bile duct by stone, tumour, traumatic stricture and primary pancreatitis, as well as enlargements associated with complete atresia of the common duct in infants, and other cases of dilatation of the cystic and hepatic ducts only.

Much of the older literature has been reviewed again, and I am in agreement with the statements of Zinniger and Cash, except as mentioned above. All literature since 1930 has been examined carefully, with the result that a number of fresh cases have been added to the list given by Zinniger and Cash (see Table II), and the references have been added to the bibliography quoted by them. They did not include cases reported by di Natale,[22] Jauregui,[23] Olivecrona,[24] Sieber,[25] Stoney,[26] and Zaczek,[27] as the literature was not available;

Gross[85] has checked these and considers the first four admissable; but, as no details are available, they have not been included in the present summary.

One case referred to by McWhorter and reported by Exner[96] in the *Wiener klinische Wochenschrift*, Volume xxiv, 1911, page 1615, could not be verified, although it is included by some authors.

Sénèque and Tailhefer in their 82 cases included cases reported by Flechtenmacher,[5] two by Keyrovsky,[88] Rosenberg,[89] Elischer,[90] Söderlund,[2] Derwissien[91] and a second case by Morley, which is that of Ashby and Platt. In agreement with Zinniger and Cash, these have been excluded from the list of proven cases with the exception of Ashby and Platt's cases.

Gross, in dealing with the condition in children, discusses only 52 cases and mentions cases reported by Badile,[104] Choyce (quoted by Stoney[26]), Erdely (two cases),[105] Ladd (two cases),[106] Morley (second case),[93] Morrin (quoted by Stoney[26]), Terrada and Vagi,[107] and Young.[108]

Morley's second case is again that attributed to Ashby and Platt. The two cases reported by Ladd are attributed by him to atresia of the common duct and should be excluded. Literature concerning the other cases has not been available, and as Gross gives no details of most of them and as the details given with one case of Erdely and that of Terrada and Vagi are not sufficient for a confirmatory opinion, they have all been omitted from this review. From brief details given by Yotuyanagi the case of Terrada and Vagi seems to be one of atresia of the common duct, as the child was only four and a half months of age and had persistent jaundice since the second week after birth.

Duff[109] reported a case in 1933 in a woman of sixty-two years who had a history of "gastritis" for seven years previously. The evidence seems very doubtful, and suggests a condition similar to that present in Reel and Burrell's case, though true bile was found in Duff's case. This case has therefore been excluded.

Wright[68] states that he has met with four cases diagnosed radiologically, and quotes one; but as the patient was not operated upon, the diagnosis could not be confirmed and the case has not been included.

Yotuyanagi[84] has written a most comprehensive article, and lists 163 references, many of which are not available to me. Altogether he mentions 55 cases, all from Japan, of which only three have appeared previously in the literature, namely, the case reported by Mayesima and two by Yamanouti. Many have but scant detail and others seem very doubtful. The three cases recorded by him are definite and have been included in the present review; but until such time as his other records can be checked, it was thought inadvisable to include any of them, although several seem undoubted.

COMMENT ON THE CASE REPORTED BY REEL AND BURRELL.

The case reported by Reel and Burrell,[38] and accepted until now, should be ruled out as being an incorrect diagnosis for the following reasons:

1. The woman was aged fifty-six years, and 90% of reported cases occur in patients under thirty years of age.

2. She had no symptoms until forty-eight years of age, and in most cases symptoms appear at an early age and have usually been present since childhood.

3. The cystic content of eight litres was at least four times greater than in any other case reported and in which the patient survived.

4. The fluid was a thin greeny-yellow and apparently not true bile.

5. Although external drainage was established, a large amount of "cystic fluid" was discharged for three weeks only.

6. An abscess subsequently developed and, when incised, drained "straw coloured fluid" for eight months before it healed. No bile is reported.

7. The patient remained well for eighteen months, and the authors note "the uneventful convalescence was an agreeable surprise".

8. Sections of the cyst wall showed islets of liver substance and dilated bile ducts in it, and the authors state "apparently it had its origin in the liver or had encroached on liver substance". No mention is made as to the cystic or hepatic ducts, nor the position of the duodenum.

9. Only two patients recovered out of 29 in whom drainage alone was used. In McConnell's case drainage was established twice, bile drained profusely and healing did not occur for four years. After that the patient continued to have symptoms, whereas in Reel and Burrell's case healing occurred quickly and the patient was well one and a half years later. Clairmont's patient recovered, but had a biliary fistula and died three years later from pulmonary tuberculosis.

10. The largest lesions reported have all been discovered *post mortem*, and Yotuyanagi's 5,200 cubic centimetres is the largest cystic content in an undoubted case, the patient being *in extremis* when seen and the condition diagnosed at autopsy.

ÆTIOLOGY.

The cause of cystic dilatation of the common bile duct remains obscure, though most authors think that a congenital factor is present. Various authors considered the following causes as productive of the condition.

1. Congenital malformation (Giezendanner,[43] Heiliger,[12] Krabbel,[50] Schürholz,[51] and Seeliger[34]). In this connexion the case reported by Heiliger[12] has been repeatedly cited; in this instance the cystic dilatation was present in a stillborn fœtus, born nearly at term. There was no jaundice, bile was present in the intestine, and the common duct below the dilatation was patent though considerably narrowed; several other congenital abnormalities were present. The onset in childhood and the occurrence of symptoms early in life, or their intermittent occurrence after beginning at an early age are evidences in favour of a congenital origin. Giezendanner[43] and Ladd[106] cited many cases of frankly congenital atresia of the common duct. In the case reported by Zinniger and Cash the thick wall of the cyst with well developed muscle and the absence of clinical signs of obstruction are points in favour of a congenital origin, as in the case now reported and in many of the others recorded. In many cases an anatomical obstruction could not be demonstrated, and in those in which obstruction could be shown, owing to kink, valve or stenosis, the dilatation above was spherical instead of the usual cylindric dilatation observed in partial or complete obstruction due to stone, tumour or traumatic stricture. Furthermore, in spite of normal walls, the gall-bladders were seldom enlarged or distended as one would expect in ordinary obstruction, and though the hepatic ducts were described as dilated in about half the cases, the dilatation was never commensurate with that of the common duct. Valve-like folds over the mouths of the cystic and hepatic ducts may have been factors in preventing the dilatation of these structures.

2. Abnormal course of the common duct or angular insertion into the duodenum, congenital in origin, which causes kinking and obstruction (Rostowzew,[58] Arnolds,[46] Konitsky,[59] Ebner,[39] Russell,[60] Schloessmann[61]). Lavenson[45] was the first to point out that this change might be

a result rather than a cause of the dilatation. We with other writers agree with this conception, according to which the progressive enlargement pushes forward or backward, and this displacement gives rise to the abnormal course. In any case, most observers note that the dilatation stops short of the duodenum, often by two or three centimetres. Kolb[62] and Dreesman[20] pointed out that the condition is unlike hydronephrosis, to which it has been compared, since in the latter the ureter normally begins as a relatively large cavity, while the common duct arises by the confluence of several small ducts and is normally of uniform calibre throughout. Furthermore, it is unlike hydronephrosis in that there is not uniform dilatation of the entire system above the obstruction (Heiliger[12]). In his article, Schloessmann[61] stated that in the majority of the cases reported (1911) an abnormality of the insertion into the duodenum was present, but this is a difficult matter to judge owing to the great distortion of normal anatomical arrangements caused by the dilatation.

3. A valve-like fold of tissue over the mouth of the intraduodenal portion of the duct (Bakes,[7] Sternberg,[8] Clairmont,[56] Neugebauer,[21] and Rostowzew[58]). The formation of the valve was described in seventeen cases. As pointed out by Lavenson,[45] Schloessmann[61] and others, it is hard to imagine how a valve-like fold could occur spontaneously in a cylindric structure and cause its dilatation into a cavity. Once a cavity is present it can easily be seen that a valve-like fold might develop as a result of sagging at the junction of the cavity with a smaller tube draining from it. The fact that such folds were present over the mouths of the hepatic and cystic ducts in many of the specimens is in favour of such an explanation. Once a valve has formed it can be seen how it might function to cause further enlargement and obstructive symptoms. That valvular action is often present has been demonstrated in a number of cases by the presence of intermittent jaundice and pain and by fluctuations in the size of the tumour under observation.

4. Congenital narrowing of the intraduodenal portion of the duct (Heid[11]) or stenosis due to infection (Edgeworth[63]). If this condition is present the lesion should be regarded as (*a*) congenital atresia or (*b*) secondary dilatation and therefore not true idiopathic dilatation.

5. Congenital weakness of the wall of the duct. This condition was first suggested as a cause by Dreesman[20] (1908), and later by Lavenson,[45] Mayesima,[36] Neugebauer,[21] Kolb,[62] Weiss,[64] Bolle[47] and Russell.[60] Several of these authors assumed an additional factor such as partial stenosis or occlusion due to catarrhal inflammation, spasm or œdema. This whole assumption, like most of the explanations that have been offered, is not easily capable of proof. In his case Mayesima[36] said of it that "no other cause was present". In the cases reported in which measurements of the thickness of the wall were given, the thinnest was 2·0 millimetres, the thickest 7·5 millimetres. In many cases the wall was described as thin but tough; in only four cases was it very thin and in two of these it ruptured during operative manipulation; in a few cases it was described as thick. Neugebauer[21] compared the dilatation to that of the colon in Hirschsprung's disease and an analogy to other achalasias has been made, but there is no record of hypertrophy or blockage of the sphincter of Oddi and in most post-mortem cases the duct is obviously patent. Also Swartley and Weeder's case (93, Table II), in which the dilatation occurred in one of the ducts only, seems to rule out this idea; as do the facts that the duct for 1·0 to 3·0 centimetres above Oddi's sphincter is not dilated and the dilatation seldom extends up into the hepatic ducts.

6. A complication of pregnancy. In some 10% of cases the symptoms bore a more or less direct relationship to pregnancy. Goldammer[29] thought that pressure from the enlarging uterus might have been a factor in his case. McWhorter[18] expressed the belief that delivery might cause a kink in the common duct due to the release of intraabdominal pressure. However, the majority of cases have occurred independently of such a possibility and many have occurred as well in the male.

7. A primary congenital condition, but with one or more of several possible modifying factors, such as infection, spasm, kink, the formation of valves or partial atresia, which cause further development of the condition (Zipf[54]).

8. Trauma. This possible ætiological factor was mentioned by Kremer.[10] In the case reported by him and by Hildebrand the patient had a fall at the age of seven or eight years. At that time the abdomen was injured and the patient herself considered that this was the cause of the symptoms. It is unlikely that trauma can play any part in ætiology.

9. Pancreatic adenomata. In his case, Budde[49] found a minute diverticulum of the ampulla of Vater and areas of fibroadenoma in the wall of the cyst which he demonstrated by serial section to be continuous with the pancreas. He described these areas as pancreatic adenomata. He believed that just as diverticula of the duodenum are present in association with aberrant pancreatic tissue, so cystic dilatations or diverticula of the common duct may be conditioned by pancreatic adenomata. In none of the other cases was fibroadenoma found. Reel and Burrell[38] found a few structures resembling bile ducts in the wall of the cyst in their case which, for reasons mentioned elsewhere, is not regarded as a case of true idiopathic dilatation. They interpreted these structures as remnants of liver tissue and thought that the cyst might be of hepatic origin.

10. Pressure on the common duct by enlarged tuberculous mesenteric glands. This condition was suggested by Douglas,[65] but has not been noted by other writers.

From a glance at the foregoing paragraphs it is readily seen that most authors regarded the condition as a congenital anomaly, though many others described it as idiopathic. In four cases in which an abdominal operation had been carried out at some time previous to the development of symptoms, the presence of the cyst was not discovered at the first operation (in the cases of Willis,[66] McWhorter,[18] Wilson,[67] and Sénèque and Tailhefer[16]), but this does not necessarily mean that a cyst was not present at that time, as in each instance the first operation was performed on the lower part of the abdomen.

Yotuyanagi has discussed the question of ætiology *in extenso* and expresses the following views:

1. The condition occurs predominantly in children or young adults. In 80% of recorded cases the patients are under twenty-five years of age, and symptoms date back for long periods as a rule.

2. It occurs in females in 76% of cases.

3. He points out that when any blockage or compression of the lower end of the common bile duct occurs, the proximal biliary ducts dilate equally, and the common duct is seldom seen larger than three centimetres in diameter, that is, the whole biliary tract shares in the dilatation. In the condition under discussion the dilatation does not involve the lower two or three centimetres of the common duct and the greatest dilatation occurs in this duct itself. Thus the two conditions differ materially.

4. He quotes several cases, including Heiliger's, indicating that the condition is congenital in origin and a primary dilatation rather than

secondary to some weakness of the duct wall. The subsequent increase in size is due to bile stagnation which is aggravated if infection occurs. This is likely to cause kinking of the duct below the dilatation, which is thereby increased and possibly accentuated by the formation of the valve-like folds described, and which may be formed by dragging of the mucosa due to increasing dilatation. Thus a vicious circle is set up.

Yotuyanagi postulates two essentials: (*a*) a congenital cystic dilatation to some degree, (*b*) a narrowing or stenosis of the terminal portion of the duct, but the latter may also be induced by the pull of the dilated portion stretching the lower part of the duct so that the usual 1·0 centimetre of the intramural part may be stretched to, say, 3·0 centimetres, with consequent narrowing of its lumen. In many cases this appearance is present, but numbers also show no stenosis but a free channel, so that dilatation *plus* stagnation of inspissated (and often infected) bile is the reason why the "cyst" does not empty itself into the duodenum. Until such time as actual obstruction to the bile flow occurs or until infection becomes a feature, the condition remains symptomless, and the only sign which may present is that of a cystic tumour in the upper part of the abdomen on the right side.

Several writers have noted the presence of this cystic dilatation with, apparently, atresia of the lower end of the duct, but others have suggested a relationship between atresia and cystic dilatation. For example, Feyrter classed them together as "malformation of the extra-hepatic biliary tract accompanied by disturbance in the formation of their normal cavity".

Hauser and Yotuyanagi agree with him, but if the congenital atresia were an important or even a constant factor, symptoms in all cases should be evident within a few months of birth, whereas in the majority of cases no symptoms are manifest for years. Therefore, one cannot agree with Yotuyanagi's conclusion that "both diseases are related intimately to each other, are essentially the same from a pathological-anatomical point of view and accordingly result from the same cause".

Yotuyanagi discusses the embryology of the area, but as this is concerned mainly with conditions of atresia, it will not be considered here. An exception must be made with one significant point that he makes which may provide a rational explanation of why congenital cystic dilatation of the duct does develop, namely that its ætiology lies in the inequality of proliferation of the epithelial cells at the stage when the primitive choledochus is still solid, perhaps at the stage of the physiological epithelial occlusion. If proliferation of the epithelial cells in the proximal portion of the duct occurs to excess, this part of the duct will be abnormally wide when canalization occurs later and the distal part relatively narrow, or if proliferation is below the normal, then actual atresia may occur to some degree or other. This is the only possible developmental relationship that can connect the two conditions of atresia and dilatation of the common bile duct; but although well argued by Yotuyanagi, the hypothesis hardly seems sufficient to determine the direct relationship he deduces. The chief value of his arguments lies in his insistence that the condition is a congenital one in the first place.

SUMMARY.

1. A case of congenital cystic dilatation of the common bile duct is recorded.

2. The article by Zinniger and Cash has been used as a basis for the present discussion. Most of their recorded cases have been reviewed, corrections have been made and material information has been added.

3. A short presentation of the salient features of the condition has been made.

4. A check review of the literature has been made in all cases in which it has been available, and the bibliography has been extended to include any articles met with since 1930.

5. Twenty-three additional cases have been included in the records.

6. Reasons for the exclusion of Reel and Burrell's case are given.

7. The ætiology has been discussed and additions have been made to the suggested causes.

8. In Table I the results of treatment have been recast and additions have been made.

9. Results of treatment show that a primary choledocho-duodenostomy is the operation of choice, but an adequate stoma must be provided.

10. Drainage with secondary anatomosis may be a life-saving measure in debilitated patients.

11. The necessity for the administration of bile salts and vitamin *K* is noted during the period of drainage.

12. A brief summary of 105 cases is listed.

13. A full bibliography is appended.

ACKNOWLEDGEMENT.

I wish to express my indebtedness to Sir Robert Wade for his correct diagnosis and interest in the case reported, and to Miss Clarke, of the Prince Henry Hospital library, for her help in securing references and original articles and for checking the bibliography.

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THE SURGERY OF PETROSITIS.[1]

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THE object of this paper is to introduce the subject for discussion this evening, to refresh your memories on some of the salient features of infection of the petrous pyramid, and to outline the surgical procedures which have been used in treatment, with some of the reasons for their origin and their use.

HISTORICAL.

A number of otologists in the latter part of the last and the early part of this century drained abscesses of the petrous temporal bone by following up fistulæ accidentally found at operation on the mastoid or middle ear.

Others reached abscesses in the petrous apex by removing portion of the squamous temporal, the *tegmen tympani* and *tegmen antri*, and portion of the bony external auditory canal, then retracting the temporo-sphenoidal lobe after separating the dura from the upper surface of the petrous bone. This operation was later used by Eagleton,[4] was renamed in 1929 "unlocking of the petrous pyramid", and is now commonly known in association with his name. In 1927, Richards[34] recorded the use of an approach to the petrous apex after destruction of the labyrinth. In 1930, Kopetsky and Almour[19] described their technique for burring a hole into the apex between the cochlea, the Eustachian tube and the internal carotid artery.

Frenckner[7] in 1932 suggested perforation by curettage through the arch of the superior vertical semicircular canal.

In 1933, Ramadier[33] recorded use of drainage through the posterior wall of the carotid canal. In 1936, Eagleton[3] suggested, and Myerson, Blumberg and Rubin[28] actually described, an operation for a lateral cervical and retropharyngeal route of approach, a procedure which rightly did not achieve any popularity. In 1936 and 1937, Lempert[24] described his complete apicectomy by exposing the internal carotid in the whole of its intrapetrous course.

ANATOMY.

The petrous portion of the temporal bone houses the membranous labyrinth, surrounding which is the bony labyrinth or otic capsule, composed of ivory-like bone which has practically reached its normal adult size at birth (see Figure I). Surrounding the otic capsule and taking the shape of the petrous pyramid is spongy bone which can be arbitrarily divided into a basal perilabyrinthine portion and an apical pericarotid portion medial to the former. This spongy bone lying partly superior, but mostly posterior, to the transverse course of the carotid canal contains marrow spaces; but may become pneumatized from the tympanic mucosa in the same way that the mastoid process becomes pneumatized, but less frequently and very often only partially. Pneumatization is normally complete in the mastoid by the fourth year of life; it is not yet established at which age normal pneumatization, when it occurs, is completed in the petrous temporal bone; it probably varies considerably in different subjects.

[1] Read at a meeting of the Royal Australasian College of Surgeons on October 12, 1939, and accepted for publication on August 7, 1940.

Pneumatization takes place by four main routes, although it is subject to many variations and combinations. The routes are: (*a*) tubal or pericarotid, (*b*) hypotympanic, (*c*) epitympanic, (*d*) antral.

The hypotympanic group of cells passes beneath the labyrinth and the epitympanic group. The cells of the antral group usually connect up with those of the epitympanic group, but may pneumatize the solid angle between the semicircular canals without further extension toward the apex. Occasionally a tract of cells may pass through the arch of the superior vertical semicircular canal, and sometimes a tier of cells is found between the posterior vertical semicircular canal and the cerebellar wall, surrounding the endolymphatic duct.

The petrous pyramid can be regarded as having four surfaces: (*a*) the posterior fossa surface, (*b*) the superior middle fossa surface, (*c*) the antero-lateral or basal tympanic surface, (*d*) the inferior skull base surface.

FIGURE I. A semi-schematic representation of the ear and its position in the skull, seen from above. Modified from Eckert-Neöbias in the Denker-Kahler *"Handbuch der Hals-, Nasen und Ohrenheilkunde"*, Volume vi. (After S. R. Guild.[14])

The geometric apex is received into the angle between the posterior border of the great wing of the sphenoid bone and the basilar part of the occipital bone.

Many important structures are concentrated in this region; for example, (i) the membranous labyrinth; (ii) the fifth, sixth, seventh, eighth, ninth, tenth and eleventh nerves; (iii) the internal carotid artery; (iv) the Eustachian tube; (v) the four petrosal nerves.

The petrous portion of the temporal bone differs from the mastoid process by the fact that many more of these important anatomical structures actually pass through it.

PATHOLOGY.

Lesions may take the form of: (*a*) osteomyelitis; (*b*) coalescent osteitis, such as occurs in infected pneumatic mastoids; (*c*) a condition developing into empyema which may discharge its contents through a fistula.

Resolution occurs in the usual way by fibrosis.

Ramadier[33] thinks that two forms of osteomyelitis are encountered. The first is hæmatogenic (similar to Eagleton's[6] acute septicæmic). This occurs

in the very young. The picture is one of a general infection with marked prostration, gastro-enteritic disturbances, and often pulmonary involvement, overshadowing the otitic signs. Infection finally reaches the lepto-meninges. The disease is usually bilateral. Ramadier's second form is otogenic osteomyelitis; it is not confined to infants and children, and often is combined with coalescent osteitis, to which it may be secondary when the infection spreads through the bony walls of the air cells to adjacent marrow spaces of non-pneumatized areas.

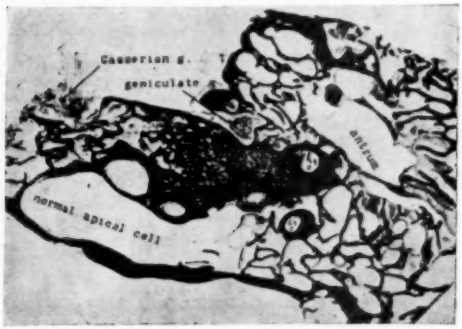

FIGURE II. Horizontal section through the petrous bone at a superior level, showing a normal apical cell in the presence of involved perilabyrinthine cells. (After Friedman.[8])

The final result of these two is much the same; that is, a coalescent abscess.

In these coalescent osteitic lesions it is important from the point of view of treatment to recognize three stages[19] (see Figures II and III): (*a*) the oto-mastoidal stage, in which infection of air spaces occurs in mastoid and middle ear regions; (*b*) the intrapetrosal stage, in which extension of infection takes place to air cells of petrous pyramid and sometimes further to marrow spaces adjacent to these cells; (*c*) the peripetrosal stage, in which the peripetrosal venous sinuses, various nerves, notably the fifth and sixth, the leptomeninges, and the naso-pharyngeal region may be involved.

THE CLINICAL PICTURE.

In the typical picture the following symptoms and signs are found: (i) neuralgic pain, sometimes hemicranial, sometimes temporal, or vaguely deep-seated behind the eye and worse at night; (ii) aural discharge or discharge from the mastoid wound, which may be recurrent or continuous; (iii) a low grade of sepsis; (iv) often a sixth nerve paralysis (see Figure IV); (v) X-ray findings showing destructive changes in the petrous pyramid, especially the apex.

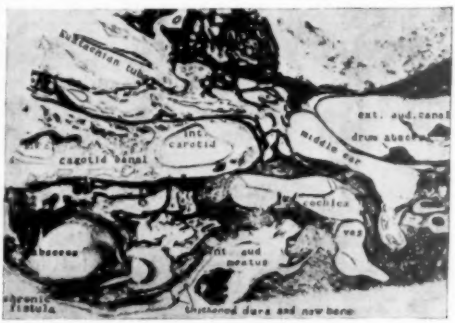

FIGURE III. Horizontal section through the petrous bone at an inferior level, showing a coalescent apical abscess connecting with the extradural space of posterior fossa by a fistula. Note thickened dura and new-formed bone in internal auditory meatus. (After Friedman.[8])

The clinical picture is often atypical and the course of the infection is often silent. Time does not allow of review of all the variations in the clinical picture this evening.

Kopetsky[20][21] divides the clinical course into four periods in typical cases: (i) the period of aural discharge, pain in the eye, mostly at night, possibly with sixth nerve paralysis, which merges into (ii) a period of low-grade sepsis, with intermittent attacks of pain; (iii) a period of quiescence with only fever, in which the pain disappears or alters

in character following perforation of the bony petrous shell; (iv) the terminal stage. In this the complications are: meningitis, facial paralysis, deafness due to involvement of the internal auditory meatus or the labyrinth, abscess of the brain. Of the sufferers from this condition 20% to 30% have died from complications in the past.

RÖNTGENOGRAPHY.

X rays have their limitations in this condition, and they are due to: (*a*) variations in anatomy; (*b*) difficulty in getting the necessary clarity of detail because of the depth of the petrous pyramid in the skull, and the necessity for minimizing the effect of the shadow of the overlying bones; (*c*) difficulty in interpreting the films—suspicious-looking lesions are encountered which are not due to infection and *vice versa*.

They may nevertheless be very useful in giving anatomical data about the type of petrous process, the extent and character of the destruction and its progress. Like X rays in mastoiditis, they are an adjunct to the clinical findings.

FIGURE IV. Section through the apex, showing very slight exudate about the sixth nerve. VIN indicates the sixth nerve; CA, the carotid artery, and E, exudate. (After Friesner and Druss.[101])

X rays may give an indication of whether or not the bony confines are intact. It is fairly easy to demonstrate the presence of an apical lesion, but it is not so easy to prove a perilabyrinthine lesion.

Three different positions are commonly used to demonstrate lesions in the apex: (*a*) the base-plate position (Taylor[38]); (*b*) the Stenvers position (oblique, taken either on face or in the recumbent position); (*c*) the antero-posterior position with the petrous apex seen through the orbit. Each is useful according to the type of case.

TREATMENT.

Treatment of petrositis may be surgical if the infection becomes a suppurative one. Not all otologists will agree on the surgical technique to be adopted, but considerable advances in the surgical treatment of petrositis have been made in the last decade and a voluminous literature on the subject has appeared.

From the point of view of treatment, petrositis may be arbitrarily divided into the following groups which have been modified from Lempert:[24] (*a*) congestive petrositis; (*b*) suppurative labyrinthitis; (*c*) suppurative perilabyrinthitis with fistulæ through the surfaces of the petrous temporal bone, either into the middle ear or mastoid antrum, or with the production of extradural abscesses; (*d*) suppurative apicitis, with or without fistulæ through one of the three apical surfaces; (*e*) a combination of suppurative apicitis and suppurative perilabyrinthitis.

Petrositis is a complication of *otitis media* in the same way as mastoiditis and may occur without mastoiditis, though not usually. The unwary surgeon in such cases, on finding a mastoid practically normal, may be led to delay further surgical relief of the petrous bone.

Here, as elsewhere, there are two objects of any surgical therapy:[21] (i) to eradicate the focus, (ii) to prevent extension of the disease.

Congestive Petrositis.

In mastoid disease, although only a small part may be infected, modern otologists remove the whole of the pneumatized process. In the petrous pyramid it is necessary to localize the area infected if possible, in order to prevent sacrifice of adjacent important functioning structures.

A number of cases of acute purulent *otitis media* is accompanied by one or more of the signs and symptoms associated with petrositis, suggesting that the infection has spread to involve some part of the petrous pyramid; but these symptoms and signs quickly disappear following myringotomy or a simple Schwartze mastoidectomy. These are cases of congestive petrositis which have resolved before the suppurative stage is reached, and as X-ray evidence does not appear until some seven to ten days after suppuration has supervened, these cases are not demonstrable radiologically.

It is conceivable that suppuration may supervene in an infection of the cells in a pneumatized petrous bone, and that the infection may be overcome just as this stage is reached. As all these cells are in ultimate communication with the tympanum, provided that there is no block caused by swollen mucous membrane or by granulations, the pus formed may drain into the tympanum, and resolution may occur without further surgical operation than either a myringotomy or a simple mastoidectomy. Drainage must also take place in some cases of mastoiditis, especially in this era of chemotherapy with the sulphanilamide preparations.

Suppurative Labyrinthitis.

Suppurative labyrinthitis is a relatively infrequent complication of petrositis and it is not proposed to include it in the discussion this evening.

Suppurative Perilabyrinthitis.

The purulent contents of the cells involved may rupture through fistulæ into the mastoid antrum or tympanum, or through the superior or posterior surfaces of the basal labyrinthine petrous into the middle or posterior fossæ, to form extradural abscesses.

The treatment is to remove the mastoid process, to expose the basal labyrinthine portion of the petrous pyramid in order to search for fistulous tracts or extradural collections of pus. All surgeons agree on careful curettage and widening of the fistula when found and, if possible, removal of the cells concerned after adequate access has been obtained. Lempert[24] maintains that all the surfaces of the basal labyrinthine portion of the petrous should be inspected and explored, as he feels that the presence of a fistula in one of the surfaces is no guarantee that there are not additional fistulæ on some other surfaces.

The writer feels that Lempert means this statement as a generalization. Unless circumstances indicated such action—for example, if perforation of the bony petrous shell was suspected—the writer would be prepared to wait before sacrificing the middle ear to explore the anterior perilabyrinthine space.

If on following up one of these fistulæ it is found to extend beyond the perilabyrinth into the apical carotid portion of the petrous pyramid, Lempert thinks that one of the procedures to be described under the treatment of apicitis will be necessary.

The lesions confined to the posterior perilabyrinth (Almour[1]) are the easiest to manage and the milder cases respond to a thorough simple mastoidectomy. When the postero-superior group of cells are affected a fistula

is found in the solid angle above the horizontal canal or more rarely through its arch.

The postero-inferior group of cells rupture through below the posterior vertical semicircular canal. The postero-internal group of cells extend toward the internal auditory meatus, and infection in them threatens an early meningitis by rupture into the posterior fossa.

In treatment of the latter, the dura of the sinus and of the anterior cerebellar surface is exposed and carefully separated toward the internal auditory meatus to expose any extradural collection. In the removal of diseased cells from the posterior perilabyrinth, which may include skeletonizing of the semicircular canals, the presence of the vestibular duct containing the endolymphatic duct and sac must be remembered. Injury to it may cause labyrinthitis or Ménière's symptom complex from interference with the drainage of the endolymph.

Lesions affecting the anterior perilabyrinth are more serious and more dangerous because they are not demonstrable through the exposure afforded by the simple mastoidectomy, and because of this the search for the focus is often delayed (Almour[1]).

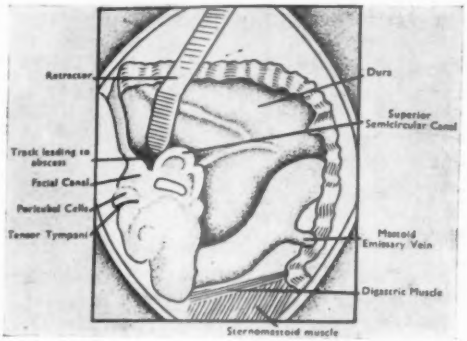

FIGURE V. Shows the left radical mastoid cavity with the track to the apex anterior to the superior vertical canal exposed. This area can be reached by extending the simple mastoid operation without removing the drum and ossicles. (After G. H. Bateman.[2])

If the anterior supracochlear group of cells is infected, the exposure afforded by a simple mastoidectomy is sufficient if the operation is extended to expose the area in front of the superior semicircular canal[2] (see Figure V). Fistulæ here often lead on into the apex, necessitating appropriate measures. The anterior subcochlear and the anterior precochlear (pericarotid) groups of cells when involved require opening of the tympanic cavity for acess.

Apicitis.

The surgical approaches to the petrous apex in apicitis may be grouped under extra petrosal (subdural) and intrapetrosal headings.

Extrapetrosal Approaches.—In the extrapetrosal procedures the dura of the middle cranial fossa is separated from the superior surface of the petrous bone (*a*) through the simple mastoidectomy exposure, (*b*) by Eagleton's[4] "unlocking" operation, or (*c*) by the Gasserian ganglion approach.

The advantages of these methods are exposure of the superior surface of the petrous for surgical attack and the drainage of an extradural collection on that surface.

The disadvantages are that posterior perilabyrinthine lesions are not adequately drained, nor are extradural abscesses in the posterior fossa.

Subdural approaches through the simple mastoidectomy, radical mastoidectomy being avoided, are inadequate and possibly dangerous procedures because the operator cannot look for anterior perilabyrinthine fistulæ. Exceptions are when the lesion is near to the superior surface of the petrous near the base or when an extradural abscess has formed on that surface. In addition, the elevation of the dura must be started behind the

superior semicircular canal and the dural insertions into the canals for the petrosal nerves and sometimes the insertion into the subarcuate fossa are torn, and many vascular connexions between the dura and petrous bone are broken, the risk of meningitis being thus increased. This risk is lessened by sealing these small openings to the subarachnoid space by diathermy. Care must be taken to avoid, if possible, injury to the greater superficial petrosal nerve, as facial paresis often follows injury.

The actual cause of this occasional facial paralysis has not been settled. In those cases in which recovery occurs after four to eight months, and probably also in transient pareses clearing up in a few days or weeks, the lesion is similar to a Bell's palsy. Infection enters by the *hiatus canalis facialis* and passes to the facial nerve at the geniculate ganglion in its bony canal. Swelling and œdema occurring in this semi-confined space produce paralysis from pressure. Another possible factor is interference with the blood supply of the ganglion from damage either to the middle meningeal itself at the *foramen spinosum* or to the small branch which enters the *hiatus canalis facialis*. This may also cause œdema with pressure on the nerve. If actual death of nerve tissue in the ganglion occurred, of course the paralysis might not completely recover, since recovery would depend on the presence of an adequate collateral circulation from the tympanic plexus.

Trophic changes also occur: dryness of the nasal mucous membranes and loss of tears due to interruption of the parasympathetic fibres to the spheno-palatine ganglion.

The subdural approach through the simple mastoidectomy is technically bad because the point of attack is further away from the objective than that of the other subdural operations (Almour[1]).

In 1934, Myerson, Rubin and Gilbert[30] described a form of subdural approach through the simple mastoidectomy exposure which they had used. A vertical incision is made from the uppermost attachment of the auricle about two and a half inches upwards, and a large section of the squamous temporal from the zygoma anteriorly to the sigmoid sinus knee posteriorly is removed, to leave an opening approximately four inches in diameter. The *tegmen antri* and the *tegmen tympani* are also removed as far as the prominence overlying the superior semicircular canal or the arcuate eminence. Elevation of the temporal lobe is first carried out along the anterior surface, the elevated area being kept as close as possible to the superior border of the pyramid. Some resistance is encountered from the arcuate eminence until the inner margin of the internal auditory meatus is reached. Beyond this lies the apical area. The surgeon uses the following anatomical markings on the antero-superior surface of the petrous as a guide to progress. First one encounters an elevation, the arcuate eminence, then a depression which posteriorly corresponds to the region of the subarcuate fossa, then a second elevation corresponding to the upper lip and roof of the internal auditory meatus, and lastly a second depression, the petrous apex. When the elevation has proceeded as far as the internal auditory meatus, the middle meningeal artery can be seen anteriorly and inferiorly emerging from the *foramen spinosum*; and a line drawn directly backward will always pass through the internal lip of the internal auditory meatus. This line serves as a guide to show the point to the medial side of which the surgeon must keep when opening the apex, which he does with a special bent gouge.

In the subdural approach through the extended radical mastoidectomy, as described by Eagleton,[4] the operator can inspect for fistulæ anteriorly; but it is very difficult to avoid the petrosal nerves and vascular connexions. This approach also does not afford dependent drainage.

Eagleton's technique is briefly as follows. The ear is reflected forward and downward. The temporal muscle, still covered by its fascia, is freed from the lower and posterior part of the temporal ridge and reflected forward and upward.

Eagleton "unlocks" the petrous pyramid by removing the "anterior and posterior buttresses". The "posterior buttress" is formed by that bone which lies in Trautmann's triangle, formed by the junction of the superior and posterior surfaces of the petrous pyramid internally, and externally where the pyramid passes into the parietal and the squama above, the mastoid process below, and the occipital behind, in the region of the asterion. The actual shape of it depends to some extent on the position of the lateral sinus. The posterior end of its root lies over the knee of the sinus.

The anterior border of the base of the petrous is locked into the base of the skull by the great wing of the sphenoid anteriorly and by the squama, zygoma, the glenoid fossa and lower jaw externally. This dove-tailed junction, made up of the tympanic ring, the medial root of the zygoma (which passes inward to form the posterior wall of the glenoid fossa), with the anterior wall of the external auditory canal, and part of the floor of the outer part of the middle fossa, is called the "anterior buttress". This buttress covers the upper and anterior two-thirds of the bony cochlea and the epitympanic space of the middle ear. It cannot be entirely removed (although Lempert removes more than Eagleton) because of its support for the jaw, but a V-shaped portion of its anterior meatal wall area can be undercut in it from behind, and its cranial surface lowered from above downward.

The *tegmen tympani* is then removed and the temporo-sphenoidal lobe elevated carefully.

The above procedure affords an exposure of the middle and posterior fossæ in their relation to the petrous pyramid, anteriorly or antero-superiorly as far as the apex, while posteriorly Eagleton goes as far as the internal auditory meatus. Difficulty is often encountered in stripping posteriorly because of the overhang of the postero-superior margin of the petrous pyramid.

The Gasserian ganglion approach by itself is not a good one because one cannot explore for fistulæ and its use presupposes that in all cases of petrositis the lesion is located in the apex (Almour[1]).

Intrapetrosal Approaches.—The intrapetrosal approaches may be classified into two groups. The first of these includes the procedures of Richards and Frenckner.

Richards[34] in 1927 recorded a number of instances in which he had surgically removed the whole of the petrous pyramid beginning with a labyrinthectomy. Some three or four of the patients suffered from cancer of the petrous, and another six from suppurative changes in the labyrinth and petrous apex, some from extradural abscesses. In most of these the condition had been neglected and considerable necrosis was present. Some of the patients had facial paralysis before operation and Richards did not attempt to save the facial nerve in several cases; in others he left a bridge of bone surrounding it. He found that the successful performance of the operation was much more difficult than he at first realized and he describes the attendant dangers and pitfalls. The modiolus of the cochlea was an especial danger, as the subarachnoid space projected far into it as the perineural sheaths of the eighth nerve; also it was prone to break off at its base, completely exposing the inner end of the internal auditory meatus with an immediate gush of cerebro-spinal fluid into the wound of such quantity as to hold up the operation completely for the time being.

Frenckner's[7] method is to curette through the arch of the superior vertical semicircular canal, passing to the apex, by going between the internal auditory canal and endolymphatic duct and sac above, and the jugular bulb and the *aqueductus cochlearis* below. This procedure in the absence of a fistula to follow is not without risk to important structures.

The second group of intrapetrosal approaches include those of Kopetsky and Almour, Ramadier, and Lempert.

These last three give exposure of all possible avenues through which an infection can enter. All three procedures are performed with a presupposition that the bony shell of the petrous pyramid is intact.

1. Almour's[19] (for it was Almour who devised the actual surgical technique) operation is conducted as follows. A simple mastoidectomy is performed and a search for fistulæ is made in the posterior perilabyrinthine region. If none is found, a radical mastoidectomy is completed and search for fistulæ is made in front and behind the superior semicircular canal.

Then the surgeon removes the anterior external auditory canal wall (without opening into the glenoid fossa) and the overhanging zygomatic root, to get a better exposure of the Eustachian orifice. The tendon of the *tensor tympani* muscle and its bony covering are removed, and the muscle itself is curetted away. Then, with a dental burr, a hole is made into the petrous tip through a pyramidal space (with its base upwards) between the cochlea, the Eustachian tube and the internal carotid artery.

The burr is entered high up on the medial wall of that part of the Eustachian tube which houses the *tensor tympani* muscle, and directly under the superior surface of the petrous pyramid (see Figure VI). It is directed at an angle of between 20° and 25° to the axis of the external auditory canal and in front of it.

After drilling for three to five millimetres, the burr is felt suddenly to enter a cavity if the apex is the seat of an empyema (see Figure VII). The burr is removed and the opening is probed with a fine blunt probe and pus wells forth. In the hands of Kopetsky and Almour there were no accidents. Other operators have recorded accidental damage to neighbouring structures, for example, the facial nerve.

Kopetsky and Almour use this technique if the usual symptom complex is found and if X-ray examination reveals intact bony margins of the petrous apex in cases in which they cannot get pus after probing a fistulous tract or if they cannot find the pathway of entrance.

If signs of an extradural abscess are present as well as of petrous tip suppuration, or if X-ray examination reveals a break in the contour of the bone of the apex, they use the subdural route.

2. Ramadier,[33] in 1933, devised a technique which consists essentially of opening into the carotid canal, after an exposure similar to that used by Almour preparatory to burring into the petrous tip; the internal carotid artery is then drawn to one side and an opening is made into the petrous apex through the now exposed medial wall of the carotid canal.

Almour maintains that both his own and Ramadier's operations keep the infection and its point of drainage within the temporal bone and away from the meninges, thus reducing to a minimum the possibility of a subsequent meningitis, and there is dependent drainage.

Mayer (quoted by Erlings) mentions the following disadvantages of Ramadier's technique. Firstly he regards the technique as difficult, necessitating careful anatomical study of temporal bones (this in itself should be no objection). Secondly, there is a danger of hæmorrhages from the internal carotid artery; this sometimes follows damage to the artery by

drain tubes and sometimes the artery is closely attached to the carotid canal and may be ruptured when an opening is made into the canal. Infection may occur, and Mayer says that the danger of the occurrence of cavernous sinus thrombosis is not imaginary.

3. In 1936 and 1937, Lempert,[24] of New York, published the details of his operation of complete apicectomy; he also gave his reasons for favouring it, and his reasons for not using the other operations already mentioned.

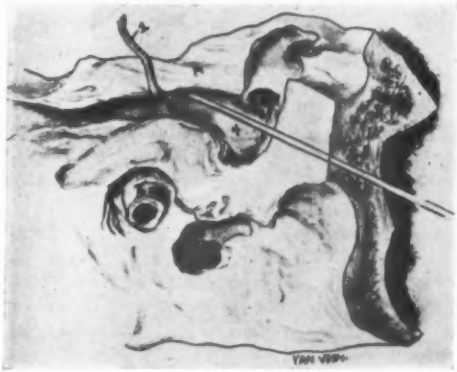

FIGURE VI. Left temporal bone prepared to show point of entrance to petrous apex. Viewed from side. The probe enters through the surgical approach to the apex. 1 = Tendon of tensor tympani left *in situ*. 2 = Tympanic orifice of Eustachian tube. 3 = Stapes. 4 = Promontory. 5 = Internal carotid artery before entering carotid canal. 6 = Jugular fossa. (After Kopetsky and Almour.[19])

He disagrees with those who think that surgery is not indicated, because in the majority recovery occurs following natural drainage through a fistula, usually situated in the Eustachian tube region; he also disagrees with those who think that operation is indicated only when the symptoms are suggestive of bacterial invasion of the subarachnoid space. He believes that such treatment constitutes poor surgical judgement. He asks does one await signs and symptoms of impending intracranial involvement before one intervenes surgically in acute suppurative mastoiditis in which the chances for intracranial complications are much more remote. His objections to Eagleton's, Almour's, and Ramadier's operations in general are that, while they do drain the pus, they do not carry out a complete exenteration of the lesion within the petrous apex, nor do they afford drainage of an extradural abscess, unless it accidentally appears in the operative field; especially is this so with those abscesses situated in the posterior fossa just internal to the internal auditory meatus. Also they do not permit of a full inspection of the entire interior of the apical carotid portion of the petrous pyramid. Lempert feels that Ramadier's operation defeats its own purpose because drainage is afforded through an opening in the internal wall of the carotid canal, which opening becomes obstructed when the carotid artery is allowed to fall back into place.

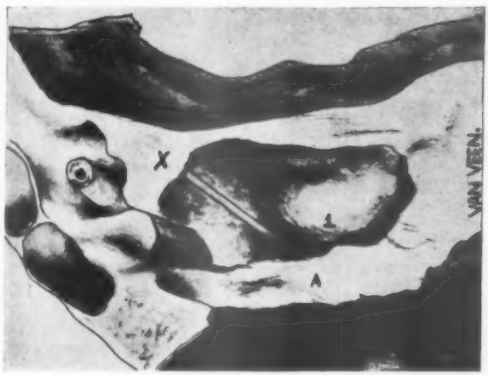

FIGURE VII. Same specimen as in Figure VI. Viewed from above. 1 = Bony cochlea. 2 = Internal carotid artery in canal. X = Petrous apex. Probe is seen to pass in groove between the cochlea and carotid artery to reach the apex of the pyramid. A = Bony overhang. (After Kopetsky and Almour.[19])

Until Lempert put forward his technique and his reasons for it, the majority of otologists had felt that when empyema of the petrous apex was present, without perforation of the bony walls of the petrous, simple drainage through a puncture as by Almour's operation was usually sufficient.

In criticism of this, Lempert disagrees with Eagleton, who states that nearly all petrositis is essentially an osteomyelitis, and as such should be treated similarly to osteomyelitis of long bones by perforation and drainage: hence Eagleton's use of the subdural approach with perforation of the apex and drainage.

Lempert agrees with Kopetsky that suppurative apicitis generally occurs in pneumatic types of apices, except in infants, and that the pneumatized petrous apex is of essentially the same structure as the pneumatized mastoid process, and also that surgery for drainage by perforation without complete exenteration is just as inadequate in the petrous as it is in the mastoid process.

FIGURE VIII. Supplementary incision for apicectomy. AB indicates the incision in the anterior wall of the external auditory canal, and BC the incision over the squamous portion of the temporal bone. (After Lempert.[21])

On analogy with the rapid healing following a modern mastoidectomy, he thinks that apicectomy by exenteration will similarly shorten the time of healing. He asks why one should expect improvement by incomplete surgical treatment in one and not the other. Adequate treatment, as by his complete apicectomy, he maintains, (*a*) affords inspection of the surfaces of the basal labyrinthine portions; (*b*) provides a means of opening the apical carotid portion and exposes for inspection the inner table formed by the posterior and inferior apical walls; (*c*) and so allows an extradural abscess in the posterior or middle fossa near the apex to be treated surgically, including treatment of a perforation of the inferior skull base surface.

Apicectomy also adequately deals with those cases in which there are multiple abscesses of the petrous pyramid, with blocking off of the connexions between the different loculi by granulations. Apicotomy may drain only the nearest one. Again the petrous pyramid may be partly sclerotic and partly pneumatic, and the drill or chisel may miss the necrotic pneumatic area.

There are four stages in Lempert's technique for complete apicectomy: (i) mastoido-tympanectomy (radical mastoidectomy); (ii) exposure and inspection of the basal labyrinthine portion of the petrous pyramid for fistulæ; (iii) creation of a surgical intrapetrosal approach to the apical carotid portion of the petrous pyramid; (iv) complete exenteration of the bony cellular structure of the apical carotid portion of the petrous pyramid, accomplished through the exploration of the entire apical course of the internal carotid artery.

In creating an approach to the apical carotid portion of the petrous pyramid, Lempert supplements the soft part incision, as in Figure VIII, to give a full view of the anterior bony wall of the external auditory canal, after displacement posteriorly of the pinna.

By removal of the posterior root of the zygoma and the *tegmen tympani* the Eustachian orifice is exposed. Half an inch of the lower border of the squama is then removed, and this allows retraction of the temporal lobe.

The peri-Eustachian tube cells and the semicanal for the *tensor tympani* with the muscle are removed. The tympanic ascending vertical portion of the internal carotid canal (see Figure IX) is next exposed by removing the tympanic plate down to the petrotympanic fissure of Glasser; this includes the anterior wall of the bony external meatus and the anterior and inferior parts of the *annulus tympanicus* and tympanic bony wall and most of the posterior non-articular part of the mandibular fossa; at the same time the projection of the parotid and soft tissues forward is compressed. By this means an additional half inch of surgical space is gained.

Now the entire tympanic ascending vertical portion of the internal carotid artery, lying in its bony canal anterior and internal to the promontory, is exposed. The horizontal portion runs along the length of the antero-superior wall of the apical carotid portion of the petrous before leaving the canal in the *foramen lacerum* at the tip. Lempert uses the artery as a guide to reach and exenterate the whole of the apex, working round it like one does with the lateral sinus in the mastoid.

In his cases he never encountered the pericarotid venous plexus in removing the bony wall of the carotid canal and no bleeding occurred. He exposes the artery mainly with a sharp curette; the wall is quite thin in the vertical part.

To obtain a clear view of the floor of the apex, it is necessary to remove some of the ivory-like bone between the cochlea and the vertical portion of the carotid; there are always about two millimetres of bone between the two, and this is removed until the floor of the apex is on a level with the jugular dome.

FIGURE IX. The upper figure shows complete exenteration of the apical carotid portion of the petrous pyramid accomplished by the complete exploration of the entire apical course of the internal carotid artery. Y' = Internal carotid artery. Z = Bony wall of posterior fossa portion of petrous pyramid. Z' = The jugular dome. The lateral sinus and the internal carotid artery as shown in the lower part of the figure are the guides to complete surgical intervention on the temporal bone. (After Lempert.[241]).

In the operation the entire antero-superior middle fossa wall of the apical carotid portion of the petrous is removed away from the dura piecemeal, injury to it thus being avoided. By this approach pericarotid collections are of course drained and fistulæ in the posterior or inferior walls of the petrous and extradural abscesses or skull base abscesses are dealt with.

In acute bacterial meningitis this operation provides a direct approach to the dura of the posterior fossa for drainage of the *cisterna pontis* and *cisterna interpeduncularis*. Since the advent of therapy with sulphanilamide preparations this procedure is very rarely found necessary.

In a case of otitic bacterial suppurative meningitis in which the immediate focus of infection has not been located previous to its onset, apicectomy is performed by Lempert for two reasons: (*a*) to explore for a possible undiagnosed focus, (*b*) to permit access to drain the *cisterna pontis*

infratentorially or the *cisterna interpeduncularis* supratentorially. He thinks that the finding of a fistula in the perilabyrinthine basal portion is not an indication to stop, but one to go on to complete apicectomy.

An additional advantage is that the seventh nerve and the promontory are never in danger of injury during apicectomy because they are both posterior to the field of operation.

In conclusion, the writer thinks that while the bony confines of the petrous pyramid are intact and a fistula is present either into the tympanum or into the neck—that is, through a non-dural surface of the pyramid—radical surgery beyond that giving free exit to the pus is not indicated immediately, unless one suspects that spread or perforation through a dural surface is imminent. Confirmation of such a suspicion will be found in a cerebro-spinal fluid cell count.

This is the attitude a surgeon may reasonably adopt when he has not seen the patient until a fistula onto a non-dural surface has already formed, because so many such patients recover completely with normal hearing.

But if the surgeon is watching a patient who as yet has given no evidence clinically of a fistula, he is in a somewhat similar position to the surgeon confronted with a case of appendicitis. He does not know onto which surface of the pyramid the pus will perforate. Because in a proportion of cases perforation will occur onto a non-dural surface, is he justified in holding his hand? If he does he will lose a proportion of patients, possibly less now than formerly, because of the advent of chemotherapy in the treatment of meningitis. The alternative is to explore when he is reasonably certain of his diagnosis. If he decides to wait a little he should watch his patient carefully and take regular cerebro-spinal fluid cell counts, and be prepared to intervene. Each case has to be treated on its own merits, and in this connexion I could do no better than quote some remarks made by Kopetsky[23] in 1938, after he had used the Lempert technique on some of his cases.

> Petrosal lesions are vastly different from each other, and no routine procedure is commonly applicable to all of them. The integrity of the cranial cavity should be protected from all hazards—even those of a surgical nature—and no lightly conceived technique should be undertaken to reach intra-petrosal parts by violating the integrity of this cavity and endangering the contents. The application of elaborate techniques to reach the petrosal pyramid through the uninfected tissue of the neck or of the pharynx, is not based on much actual experience. They may be characterized as "cadaver operations" rather than practical surgical procedures, and should not be attempted to handle any of the lesions that petrosal pyramid infections present; because all lesions can now be reached by a proper selection of the type of operation to reach the locality where the lesion is located intra-petrosally. These operations are all devised to keep the surgical field within the limits imposed by the boundaries of the temporal bone and no uninfected tissue need be touched, and no pus need be drained into sterile areas from the purulent contents of the petrosa. To do so adds extra hazards to a situation already grave enough.
>
> Finally, every otological surgeon must be competent to perform all the recognised techniques upon the petrosal pyramid, so that he may select the one suitable for a given lesion, rather than be perfect in one procedure and habitually perform it, whether or not it meets the indications presented. Surgery, of course, should be as conservative as adequacy will permit. True conservative surgery is ideal when it reaches its objective. Surgery which fails to accomplish this goal is neither conservative nor adequate. It is futile.[1]

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Surgical Technique.

PULP TRACTION: THE STILETTE METHOD.[1]

By B. T. KEON-COHEN,
Melbourne.

TRACTION is necessary in the treatment of certain fractures of metacarpals, metatarsals and phalanges. The most efficient method of securing such traction is by means of a fine stainless steel wire through the pulp of the terminal phalanx.

The following technique is extremely simple. Trauma during the introduction of the wire is negligible, and a finer wire (gauge 30) is used than in the method described by Böhler. These factors have no doubt contributed to the complete freedom, so far,

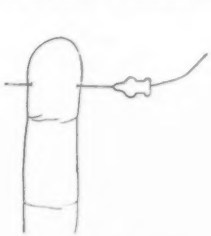

FIGURE I.

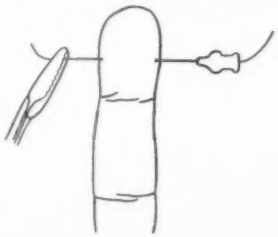

FIGURE II.

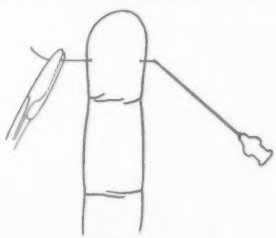

FIGURE III.

FIGURE IV.

from infection and/or wasting of the pulp, sequelæ that have occurred not infrequently following pulp traction by other techniques.

The illustrations show clearly the steps of the procedure, so that little description is required. As usual, a plaster cast is applied to the forearm or leg. Incorporated

[1] Accepted for publication on October 29, 1940.

in the cast, and extending distally so as to clear the extended fingers or toes, is a racket-shaped Cramer wire frame, or a loop of stout wire, shaped at the end like a Thomas splint.

Local anæsthesia is usually quite satisfactory. The needle selected is the smallest that will "take" the wire after the manner of a stilette.

1. Transfix the pulp of the terminal phalanx with the needle (Figure I).
2. Thread the wire through the needle like a stilette (Figure II).
3. Secure the distal end of the wire and withdraw the needle. The wire is left *in situ* (Figure III).

The spreader should be wide enough to ensure that the wire, distal to the level of transfixion, clears the side of the pulp. It is "made to measure" from an ordinary wooden tongue spatula.

Traction is obtained by means of fine rubber tubing passed proximally through a hole bored in the centre of the spreader (knotted to prevent it pulling through), and attached distally to the wire frame. The tension of the tubing is adjusted as required, so that the traction is both elastic and continuous (Figure IV). Serial X-ray pictures are essential from time to time to check the position of the fracture. In actual practice, once reduction is obtained, it is easily maintained by a minimum of tension. Whether the hand or foot is involved, the treatment is ambulatory. As in the case of all fractures, and especially in those where traction is part of the treatment, the secret of success is constant supervision.

Acknowledgement.

I wish to thank Mr. Preston, junior, Anatomy School, University of Melbourne, for Figures I, II and III, and Dr. Ket, of Prince Henry Hospital, for Figure IV.

The Australian and New Zealand Journal of Surgery.

All articles submitted for publication in this journal must be typewritten and double or treble spacing should be used. Each article should conclude with a brief summary and statement of conclusions. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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References to articles and books should be carefully checked. In a reference the following information should be given without any abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given, with full date in each instance.

When illustrations are required, good photographic prints on glossy gaslight paper should be submitted. Line drawings, charts, graphs and so forth should be drawn on thick white paper in indian ink. Authors who are not accustomed to prepare drawings of this kind, are invited to seek the advice of the Editor if they are in any doubt as to the correct procedure. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available.

VOL. XI. JULY, 1941. No. 1.

SURGERY IN THE DESERT.

IT has been said that a surgeon is a physician who can operate. Although this may be regarded by many as assuming too many virtues in the surgeon, it is undoubtedly true that the best surgeon is also a good physician. The necessity for special and what in other circumstances could be regarded as purely "medical" knowledge becomes acute when the individual is segregated by many miles from his colleagues and possibly hundreds of miles from specialist physicians.

There is a number of problems requiring not only particular knowledge, but a degree of adaptability seldom demanded in civil practice. Some of these will be mentioned, but, as far as a broad knowledge of his own subject is concerned, cases demanding the immediate differential diagnosis between a chest wound and superimposed anxiety state on the one hand and a pressure pneumothorax on the other (to mention only one condition) may, in a poorly-lit tent in inclement weather with relatively untrained assistants and minimal equipment, tax the most highly qualified practitioner. Apart, however, from purely professional problems, others arise which suggest the extreme desirability of the surgeon's being a Jack of all trades. The problems mentioned here specially are those of equipment, personnel and weather.

The question of surgical equipment of various units has given, and probably will always give, rise to considerable differences of opinion depending on the point of view of the individual—according to whether he is responsible for providing, transporting or actually using it. Surgical equipment provided with surgical teams going into forward areas has been

well thought out and is very good, but, of necessity, many items which are at hand in a general hospital, cannot always be provided. It need scarcely be mentioned that transport difficulties may be very great and always provide some difficulty so that only essential items are included in an equipment scale. Few things, however, cannot be improvised in some way. It may be of interest that three surgeons in three different places many miles apart all improvised an excellent Rehfuss tube by extracting a 0·32 bullet from its cartridge and inserting it into the end of thin rubber tubing. Lateral openings were then easily cut. Such tubes passed easily into the stomach when a simple tube would not pass the cardiac sphincter. Other examples of such improvisations could be given.

That any splint may be replaced by one contrived from plaster of Paris is well known and there is no doubt that this method of splinting has proved most valuable in these circumstances. All the surgeons working in this region have had reason to be thankful for the training in the use of this most valuable medium. At the same time it should be said that splints have appeared which have never come within our previous experience, and they have proved to be most efficient for the transport of patients in comfort.

The second problem is that of personnel. The orderlies, both in the operating theatre and in the tents, are relatively (and, compared with the hospital sisters, totally) untrained. In the early stages this means that a special watchfulness on the part of the surgeon is required, that all non-essentials (however æsthetically comforting) must be discarded, that instructions must be repeated *ad nauseam*, and, in order to allow for a long latent period, that orders be given at a much longer time before their execution is imperative. At the same time, it is often astonishing how quickly these lads learn to master relatively intricate technical manœuvres. On one occasion I was personally, to say the least, somewhat taken back to find that a corporal had refilled a "Soluvac" bottle and reset it in a tent lit only by a hurricane lamp during a severe storm. Twelve hours before he had had no experience of continuous intravenous therapy! The patient, who was desperately ill, made a good recovery. This is mentioned because it illustrates one of the features we have noticed, that the orderlies show a capacity for rising to the occasion that would not have been suspected at an earlier stage.

The third main problem is that bound up in the geographical and meteorological peculiarities of the country. Most surgeons are at first appalled at the idea of performing abdominal or cranial operations of some magnitude with only a relatively small amount of water available. It is soon appreciated that, with a little (compulsory) care, it is possible to produce sterile instruments, a clean operative field and clean hands.

The dust which percolates or drifts through every crevice of a tent was at first a cause of much anxiety. This is written with some feeling, as there is, at the time of writing, a well-developed Libyan dust storm in progress. The accumulation of dust occurs in an amount and at a rate which surpass those of most areas thought to be dusty in Australia. This has been dealt with

by an addition to the general technique which involves keeping everything covered or partly covered throughout the whole of such period. It is fortunate, however, that, as was discovered at an early period, desert dust is clinically sterile. The position was succinctly stated by a British officer who, when discussing this question, said: "The desert is kind." It is perhaps not necessary to say that this is fortunately more real than apparent—or than might be expected. The way in which improvisations will surmount most difficulties and the manner in which surgeons, faced with a particular difficulty, will apply these are well shown in this very problem. Two units, one British, the other Australian, and nearly two hundred miles apart, both employed an ingenious method of dealing with the dust. A large tent was erected and a smaller one was then pitched inside it. When there was a raging dust storm outside, there was only a very mild one in the large tent, but in the inner one, not only was one relieved of the noise, which after many hours becomes irritating and even disturbing, but the dust was reduced to a reasonable minimum capable of being readily controlled.

These few notes have been made to indicate how different the problems facing the military surgeon may be from those encountered in peace time. In a great many ways indeed the exigencies of the circumstances may cause the purely surgical considerations (as understood in normal conditions) to take second place. At the same time they develop to a high degree that capacity for dealing with almost any difficulty in most disadvantageous conditions which is obviously latent in all our personnel. This is demonstrated by similar difficulties being overcome independently by different surgeons who could not possibly have communicated with each other. It is doubtless this stimulation which is responsible for so many of the advances that arise during a war—as happened in the Great War and may well do so in the present conflict.

E. S. J. KING.

Reviews.

Rose and Carless' Manual of Surgery for Students and Practitioners. By C. P. G. WAKELEY, D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.A.C.S., F.R.A.C.S., and J. B. HUNTER, D.Sc., F.R.C.S., F.R.S.E.; Sixteenth Edition (in two volumes); 1940. London: Baillière, Tindall and Cox. Royal 8vo, pp. 1,768, with 1,090 illustrations and 19 colour plates. Price: 30s. net.

THE sixteenth edition of "Rose and Carless' Manual of Surgery", which has just been published, is a tribute to the industry and sense of proportion of the authors, Cecil Wakeley and John B. Hunter, who collaborated in editing this work. They have provided the medical student, the practitioner and the specialist surgeon with a comprehensive review of the whole field of surgery.

It is doubtful whether any other two volumes on the subject in the English language contain as much condensed, yet easily readable, matter.

Of the one thousand and ninety illustrations, many of which are in colour, three hundred are new and peculiar to this edition. Some of the old well-known familiar pictures are to be found, but the enrichment of the book by new plates and drawings indicates the manner in which the work has been brought up to date.

The early chapters are devoted to the principles of pathology and technical methods employed in surgery. Useful information on the course and treatment of burns, especially electrical, X-ray and gas, is provided. In this connexion it may be remarked that the final chapter in the book deals with war surgery, and although only sixteen pages are devoted to this special subject, much relevant matter in this field is incorporated in the appropriate sections of the main body of the work.

The well-established and time-honoured methods of wound treatment are described. Wisely enough, Trueta's method of plaster enclosure of wounded limbs is mentioned, but not unduly emphasized. Further experience in war surgery will doubtless provide the authors with new material for this important section.

The student will appreciate the concise and lucid paragraphs on dental sepsis and the necessary instructions for the performance of dental extractions.

No mention is made of the use of strapping or "Elastoplast" in the treatment of boils.

The chapter on fractures is well up to standard, and many of the recent advances in this field have been included. Spinal injuries and disease are well described and the modern ideas on sciatica are appraised. Dislocations are described in the usual manner, but the treatment of the dislocation of the shoulder of some weeks' duration is omitted.

The advocacy of "Thorotrast" injections and radiography in doubtful breast tumours is not in the old Rose and Carless tradition; time may show that this method is not free from risk. The excellent account of cancer of the breast and its treatment atones to some extent for the excess of enthusiasm for the radiological diagnosis of simple conditions.

The surgery of appendicitis is thoroughly dealt with by one who, obviously, has had long experience. This chapter might be regarded as perhaps the most valuable in the book, in view of the fact that the textbook is so generally recommended in undergraduate schools.

The recent work on the necessity for pre-operative preparation in, and the metabolic aspects of, intestinal obstruction have not received the attention they deserve.

The treatment of inguinal hernia is not so well done as we should have desired. We agree with the authors in passing briefly over the injection methods in the treatment of hernia.

Why the authors should describe at such lengths the Harris operation of prostatectomy and fail to advise the less expert in simpler methods more usually adopted by general surgeons, one cannot understand. Perhaps the modern tendency to specialization accounts for the fact that the surgery of the lower part of the urinary tract and that of the female genital organs are not so fully and expertly described.

The chapter on amputations has been enlarged. Most valuable advice is available to those who are now undertaking this work. Modern prosthetic appliances are beautifully illustrated.

A useful chapter on tropical surgery is provided, and many pictures and photographs accompany the text.

While admitting the defects as above enumerated, we find that this edition is certainly the best which has yet appeared, and it must be conceded that the many students and practitioners (old and young) who have been "brought up" on "Rose and Carless" and have an affection for the book, will welcome the publication of such excellent volumes.

The authors are to be congratulated and the more so by overseas readers, who realize that the production of any new book under the war conditions in England is no mean feat. We salute the surgeons of England.

The 1940 Year Book of Industrial and Orthopedic Surgery. Edited by C. F. PAINTER, M.D.; 1940. Chicago: The Year Book Publishers. Crown 8vo, pp. 484, with illustrations. Price $3.00 net.

THIS useful guide is the latest addition to the "Year Book" series and contains numerous epitomes, short but adequate, of traumatic and orthopædic surgical literature. Condensed accounts of the latest works published by well-known authorities appear here; illustrations are numerous; 276 pages have been selected for abstraction and 299 illustrations are reproduced on art paper. The book appears to fulfil the object for which it was designed—that is to supply in condensed form a permanent file of current literature—and can be strongly recommended for the busy man who wishes to keep abreast of this specialty.

A Short Practice of Surgery. By H. BAILEY, F.R.C.S., and R. J. M. LOVE, M.S., F.R.C.S.; Fifth Edition; 1941. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 1,023, with 880 illustrations. Price: 30s. net.

THE fifth edition of this popular book enhances the reputation it has made since it first appeared in 1932, as it has been kept well up to date both in regard to letterpress and the illustrations, which are a feature as many are in colour. Brevity and dogmatism are necessary in a work of this character, which is primarily intended for students, but must be of great value to a busy practitioner desirous of checking up on the salient points of either diagnosis or treatment in any particular case.

Small print is used for unusual conditions or special procedures or when comparisons may be made, such as in the chapter dealing with lesions of the stomach.

Bibliographical notes are short, but serve to keep fresh in the minds of the younger generation some of the men who have helped develop the art of surgery; they include even those who are contemporaries. An excellent feature is the tabulation of important details and the very neat classifications given. In most conditions some idea is given as to prognosis—a point usually neglected in such books.

The authors state that deletions and additions have been necessary in every chapter, some of which have been rewritten. That this is so seems clear from the inclusion of many recent methods in both diagnosis and treatment.

The chapter on appendicitis is a mine of valuable information, and if the advice given was followed closely, many lives would be saved. That part regarding the selection of cases for Ochsner-Sherren treatment puts the pros and cons in a fashion plain for all to understand.

Many of the aphorisms quoted are very apt and tend to fix important points in the mind, such as: "Beware of the individual with the glass eye and a large liver"; or "Purgation means perforation" *et cetera.*

This book must continue its well deserved popularity as long as the authors can keep it as up to date as the present edition. The publishers are to be congratulated upon the excellence of the paper and printing, upon the absence of typographical errors, and upon the first-rate reproduction of coloured photographs and the other illustrations.

Surgery of Modern Warfare. Edited by H. BAILEY, F.R.C.S. Part II, 1940. Edinburgh: E. and S. Livingstone. Super royal 8vo, pp. 163, with illustrations. Price: 12s. 6d. net. Part III, 1941. Edinburgh: E. and S. Livingstone. Super royal 8vo, pp. 159, with illustrations. Price 12s. 6d. net.

THE chapter on skin grafting is completed in Part II and gives a very good account of the principles involved. An interesting account of maggot therapy follows, but many technical difficulties must be overcome before this type of treatment could be applicable under war conditions. Section III deals with the wounds of the trunk, and as it numbers among its authors Tudor Edwards, Sir John Fraser, G. Gordon-Taylor and Sir Charles Gordon-Watson, it contains, as is to be expected, a great deal of wisdom and much knowledge drawn from the storehouse of experience. Injuries of blood vessels are dealt with in Section IV, and the two chapters contributed by Bruce on the exposure of the main vessels of the limbs are excellently written and illustrated. The chapter on secondary hæmorrhage is also very practical. Learmonth writes of the suture of arteries; but, as Matheson and Murray point out in their article on conservative vascular surgery, such procedures would be possible only if the patients concerned could be admitted to a fully equipped hospital within a short space of time. Nevertheless, with the aid of heparin it may yet be possible to maintain the circulation for a number of hours in a limb by the use of some form of tube joining the cut ends of the vessel, thus allowing the patient to reach a fully equipped hospital in better condition.

In Part III, Souttar writes of injuries to peripheral nerves, and Mouat and Hart of wounds of tendons. Whereas the latter chapter contains explicit instructions regarding primary and secondary suture, the former makes no mention of these distinctions and contains no advice as to when nerve suture can be undertaken with a reasonable chance of success. There follow several chapters on methods of immobilizing the limbs, including the use of the Thomas splint, the use of Thomas frames, the use of Braun's splint and Cramer wire, all of which are practical and well illustrated. Britain's chapter on methods of application of plaster of Paris is a most excellent exposition of a difficult subject. Mouat is responsible for wounds of the hand and infected wounds of the hand, and Holdsworth for wounds of the foot. Section VIII, on wounds of bones and joints, is not complete in Part III and will be reviewed with Part IV, to which we look forward at an early date.

Proceedings of the Royal Australasian College of Surgeons.

THE HYDATID REGISTRY.

THE following is the report of the Hydatid Registry of the Royal Australasian College of Surgeons for the twelve months ended December 31, 1940, submitted by the Registrar-in-Chief, Sir Louis Barnett:

The total number of cases recorded is now 1,552, with 193 deaths.

This tally of deaths is an under-estimate of the mortality, as, with few exceptions, it comprises only the deaths occurring in hospital and does not include the considerable number of cases that die in their own homes from the effects of hydatid disease. A reasonable estimate of hydatid disease mortality based on personal experience is 15%.

The records are classified and indexed for easy reference and précis cards are also on file, thus facilitating large-scale collective investigations.

During the year collective work of this kind has been carried on by the Registrar-in-Chief, and amongst other subjects dealt with are: (i) multiple abdominal and pelvic hydatid cysts, their frequency, pathogenesis, clinical features, prognosis and treatment; (ii) the distribution of hydatid cysts in different parts of the body.

Articles on these subjects have been prepared for publication in THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY.

The first record in New Zealand of a case showing hydatid lesions of the alveolaris type was added to the Registry this year. This case, similar in many respects to the unique case described by Professor H. R. Dew in THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY of September, 1931, will be fully described and discussed in the same journal at an early date.

Classification of the Cases according to the Part of the Body Chiefly Affected.

Region Affected.	Cases.	Deaths.
Liver	890	128
Multiple abdominal and pelvic, mostly secondary to liver	124	16
Lung	339	28
Muscles and fascia	74	2
Bone—		
Spine	20	3
Other bones	26	3
Kidney	36	7
Spleen	20	2
Brain	12	3
Heart	3	1
Thyreoid	2	—
Parotid	1	—
Prostate	1	—
Pancreas	1	—
Breast	2	—
Orbit	1	—
Total	1,552	193

As pointed out in the last annual report (THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, July, 1940), multiplicity of location is very common; for example, in approximately 17% of the records indexed under the caption liver cysts there are references to cysts in other parts of the body of the same patient.

Cases already in the Registry are in many instances recorded afresh because of recurrences or new cyst developments. With few exceptions these cases are filed under their original index numbers.

Follow-up notes when they come to hand (which is not often) are duly inscribed in their appropriate record forms.

The quality of the case recording by house surgeons shows some improvement, although errors and omissions, particularly omissions, are still far too frequent. Collective study of well recorded cases is an important factor in clinical progress, and Fellows of the College are again urged to take more interest in the way notes of their cases are recorded.

Hospital registrars are supposed to supervise case recording, but as a rule their chief interest, naturally enough, lies in the operating theatre, wards and out-patient departments, and their scrutiny of the records suffers accordingly. Sometimes it is very perfunctory.

The Registrar-in-Chief has sent grateful acknowledgements individually to all who have sent in their records, and special thanks are again due to Mr. H. G. Wheeler, the College Secretary, for his helpful work in stimulating surgeons and medical superintendents to send in their hydatid records, and to the local registrars in New Zealand, Messrs. J. Fitzsimons of Auckland, D. White of Wellington, and D. McK. Dickson of Christchurch.

Dr. Roland Fulton of Dunedin and the local registrars in Australia have been too busy in other ways to give much time to the rather onerous work of the Hydatid Registry, but the Registrar-in-Chief thanks them at any rate for their goodwill.

Particularly encouraging this year has been the interest shown in the Registry by Messrs. M. P. Susman (Sydney), C. J. Officer Brown (Melbourne), R. S. A. Graham (Waikato) and J. H. North (Palmerston North).

L. E. BARNETT,
Registrar-in-Chief.

ANNUAL REPORT OF THE COUNCIL.

THE Council of the College takes pleasure in submitting to Fellows the following statement recording College activities during the past twelve months.

Meetings of the Executive Committee.

The Executive Committee has met on thirteen occasions, the attendances being as follows:

Sir Hugh Devine	10	T. E. Victor Hurley (on Air Force service)	6
Sir Alan Newton (on military service)	7	A. L. Kenny	12
Balcombe Quick	13	G. R. A. Syme	8
W. A. Hailes (now on active service)	11	Sir Robert Wade	1

Meetings of the Council.

Two meetings of the Council of the College have been held since the thirteenth annual general meeting. The attendances were as follows:

Sir Hugh Devine	1	T. E. Victor Hurley (on Air Force service)	2
Sir Alan Newton (on military service)	1	A. L. Kenny	0
E. D. Ahern	1	Sir Henry Newland	1
F. Gordon Bell	0	H. R. G. Poate	2
George Bell	2	Balcombe Quick	2
H. R. Dew	1	Sir Robert Wade	2

Number of Fellows.

The College now has a fellowship of 670. This figure is made up as follows:

Honorary Fellows		17
Overseas Fellows		13
New Zealand Fellows		134
Australian Fellows—		
New South Wales	186	
Queensland	50	
South Australia	48	
Tasmania	13	
Victoria	183	
Western Australia	26	
	—	506
		670

Deaths of Fellows.

It is with regret that the Council reports the deaths of the following Fellows: Thomas Fergus (New Zealand), J. G. W. Hill (New South Wales), M. C. F. Morkane (New Zealand), A. C. Sandston (New Zealand).

Gordon Craig Library.

The books and journals available to Fellows at the Gordon Craig Library, are constantly being added to. The Council has adopted a policy of purchasing specially selected and recommended new books. A list of these additions to the library is published periodically in THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY.

The next issue of the library catalogue will be made in June, 1941.

The Council desires to inform Fellows that a most valuable gift of books and journals has been received by the library from Mr. A. L. Kenny. This gift greatly enhances the value of the ophthalmological section of the Gordon Craig Library, and members of the Council desire to record their gratitude to the donor.

In addition to this gift, the Council desires to place on record its gratitude to the following donors of books to the Gordon Craig Library: The President of the Royal College of Surgeons of England, Dr. Cowlishaw, Dr. J. W. Davis, Sir Hugh Devine, Dr. Dungan, Robert Fowler, Mr. W. Russell Grimwade, W. R. Groves, J. Bruce Hamilton, W. S. Kent Hughes, Thomas King, Dr. C. V. Mackay, F. P. Morgan, Sir Henry Newland, W. R. Nickson, G. Douglas Robb, Henry Searby, G. R. A. Syme, and Mr. C. P. G. Wakeley.

Gordon Craig Educational and Research Scholarships.

The Council of the College has undertaken to finance certain research work of national importance in connection with Australia's war effort. Grants for this purpose have already been made to the Prince Henry Hospital, Little Bay, New South Wales, and to the Walter and Eliza Hall Institute of Research in Pathology and Medicine, Victoria.

The Journal.

Some difficulty has been experienced in maintaining the standard of the journal. These difficulties are the result of circumstances associated with Australia's war effort. It is, however, expected that for the present it will be possible to publish the journal with the present quality of art paper, although at a later date this may not be possible. It is also necessary to alter the form of the journal and to use a smaller type in an endeavour to carry out the obligations of the College to the Commonwealth Government concerning economy in the use of paper. Although a journal of fewer formes will shortly appear, a rearrangement in the set-up of the journal will mean that actually more material will be published than was formerly published in a larger sized journal.

Meetings Arranged by the State and Dominion Committees.

New South Wales: Sydney, annual meeting, September 5 and 6, 1940; Sydney, annual meeting, April 3, 1941.

New Zealand: Wellington, annual meeting, September 5, 1940.

South Australia: Adelaide, annual meeting, August 14, 1940.

Tasmania: Hobart, annual meeting, September 6 and 7, 1940.

Some States have experienced, and are experiencing, difficulties in arranging their usual programme of scientific meetings.

Ryan Scholarships.

The examinations for the Michael and J. P. Ryan Scholarships in Surgery were held in December, 1940. The results were as follows: Michael Ryan Scholarship in Surgery, won by J. T. Cahill; J. P. Ryan Scholarship in Surgery, won by J. H. Lindell.

Joint Committee of the Councils of the Royal Australasian College of Surgeons and the Royal Australasian College of Physicians.

Regular meetings of the joint committee of the councils of both colleges have been held in the various States, and half-yearly joint meetings of the councils of both colleges have been arranged. At these meetings matters of common interest to both your College and the Royal Australasian College of Physicians have been given consideration.

Australian Red Cross Society.

The headquarters administrative staff of the Australian Red Cross Society continues to occupy a large section of the College building. The Council feels that, by making the unused portion of the College building available to the Australian Red Cross Society, a useful contribution to Australia's war effort is being made.

Services of the College in an Advisory Capacity.

It is pleasing to note that, with the growth and development of the College, public bodies charged with the responsibility of administering the health services of the country have, from time to time, sought the assistance and advice of the College on technical matters. In this connexion, the Hospitals Commission of New South Wales and the Charities Board of Victoria have referred problems with which they were confronted to the College for advice.

Balance Sheets and Accounts.

Copies of the balance sheets of the College have been handed to all Fellows present at this meeting, and, in due course, they will also be published in THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY. The detailed accounts are available at the meeting for inspection by Fellows and later in the meeting Fellows will be asked formally to adopt the accounts. Any information concerning the accounts will be supplied on request.

Election of Council.

Six nominations were received to fill the six vacancies on the Council. All the retiring members of the Council were renominated.

H. G. WHEELER,
Secretary.

ROYAL AUSTRALASIAN COLLEGE OF SURGEONS.

Balance Sheet as at January 31, 1941.

LIABILITIES.	£ s. d.	£ s. d.	ASSETS.	£ s. d.	£ s. d.
The Union Bank of Australia Limited—Account Overdrawn		196 4 6	Cash on Hand		30 0 0
Sundry Creditors		13 2 4	Advances to Honorary Secretaries		80 0 0
Subscriptions Paid in Advance		31 10 0	Investments—		
Examination Expenses Reserve Account		435 9 5	Syme Memorial Endowment	2,727 8 4	
Investment Fluctuation Reserve		89 16 9	T. F. Ryan Endowment	5,160 9 5	
Prince Henry's Hospital, Melbourne—Post-Graduate Teaching Fund		10 10 0	General	7,919 16 4	
Gift Account		1,538 16 9			15,807 14 1
Syme Memorial Endowment Fund	2,511 0 11		Subscriptions in Arrear		147 0 0
Syme Memorial Endowment Income Account	216 7 5		Sundry Debtors		67 7 3
		2,727 8 4	Land and Buildings—at Cost		13,992 5 11
T. F. Ryan Endowment Fund	5,100 0 0		Furniture and Fittings—at Cost—*less* Depreciation		2,218 0 0
Michael Ryan Scholarship Income Account	30 4 8		Great Mace—at Valuation		500 0 0
J. P. Ryan Scholarship Income Account	30 4 9		Craig Endowment Assets (as per separate Balance Sheet)		63,324 3 11
		5,160 9 5			
Surplus Account—					
Balance, January 31, 1940	21,573 0 6				
Add Surplus Revenue for the year ended January 31, 1941	1,065 19 3				
		22,638 19 9			
Craig Endowment Fund (as per separate Balance Sheet)		63,324 3 11			
		£96,166 11 2			£96,166 11 2

(Sgd.) H. B. DEVINE, President.
BALCOMBE QUICK, Honorary Treasurer.
H. G. WHEELER, Secretary.

Audited and found correct.
(Sgd.) YOUNG AND OUTHWAITE,
Chartered Accountants (Australia),
368 Collins Street, Melbourne.
February 11, 1941.

CRAIG ENDOWMENT.

Balance Sheet as at January 31, 1941.

LIABILITIES.	£ s. d.	£ s. d.	ASSETS.	£ s. d.
Endowment		59,056 19 2	The Bank of Australasia—Endowment Current Account	18 3 0
Investment Fluctuation Reserve		175 9 7	Endowment Investments	59,214 5 9
		59,232 8 9		59,232 8 9
Revenue Account—			The Union Bank of Australia Limited—Income Current Account	2,241 2 3
Balance, January 31, 1940	3,595 3 3		Gordon Craig Library—at Cost	1,417 12 11
Add Surplus for the year ended January 31, 1941	2,433 10 10		Library Furniture, Fittings and Equipment—at Cost—*less* Depreciation	433 0 0
	6,028 14 1			
Less Transfer to Endowment	1,936 18 11			
		4,091 15 2		
		£63,324 3 11		£63,324 3 11

(Sgd.) H. B. DEVINE, President.
BALCOMBE QUICK, Honorary Treasurer.
H. G. WHEELER, Secretary.

Audited and found correct.
(Sgd.) YOUNG AND OUTHWAITE,
Chartered Accountants (Australia),
368 Collins Street, Melbourne.
February 11, 1941.

COUNCIL MEETING.

THE Council met on Thursday, April 3, 1941, and the following matters arising out of the meeting are published for the information of Fellows:

Election of President and Vice-Presidents.

Mr. E. D. Ahern wa selected to the Presidency of the College.

Sir Alan Newton was reelected a Vice-President of the College and Mr. H. R. G. Poate was elected to the office of a Vice-President.

Election of Council.

Six nominations were received for the six vacancies on the Council. All the retiring members of the Council were renominated and declared reelected. The members of the Council reelected are as follows: F. Gordon Bell, Sir Hugh Devine, H. R. Dew, Sir Alan Newton, H. R. G. Poate, Sir Robert Wade.

Appointment of Executive Committee.

The following members of the Council were appointed an Executive Committee: Sir Hugh Devine (Chairman), Sir Alan Newton, T. E. Victor Hurley, A. L. Kenny, Balcombe Quick.

Appointment of State and Dominion Committees.

The Council passed the following resolution concerning the appointment of State and Dominion committees:

> That the Council expresses the opinion that appointees to the State and Dominion committees to fill vacancies caused by the absence on active service of members of those committees should be asked voluntarily to retire on the return from active service of the men for whom they are acting as deputies.

The following appointments were made:

New South Wales: A. J. Aspinall, W. T. D. Maxwell (as deputy for V. M. Coppleson), F. Brown Craig, B. T. Edye, J. W. S. Laidley, A. M. McIntosh, E. M. Fisher (as deputy for I. Douglas Miller).

Queensland: J. C. Hemsley, A. E. Lee, H. S. McLelland, J. J. Power, Neville G. Sutton, Hedley J. Brown.

South Australia: T. A. Britten Jones (as deputy for L. C. E. Lindon), R. M. Glynn, I. B. Jose, R. E. Magarey (as deputy for B. H. Swift), P. S. Messent, L. A. Wilson.

Tasmania: F. W. Fay, B. Hiller (as deputy for J. Bruce Hamilton), H. W. Sweetnam (as deputy for D. W. L. Parker).

Victoria: A. Fay Maclure, J. Newman Morris, Henry Searby, C. Gordon Shaw, John H. Shaw, G. R. A. Syme (as deputy for W. A. Hailes), B. T. Zwar.

Western Australia: J. P. Ainslie (as deputy for F. J. Clark), H. B. Gill, D. D. Paton.

New Zealand: Sir Carrick Robertson, D. S. Wylie, David Whyte, Sir Hugh Acland, P. Stanley Foster, J. A. Jenkins.

Appointment of State and Dominion Hospital Committees.

The following appointments were made:

New South Wales: A. J. Aspinall, A. S. D. Barton, T. Hamilton, C. B. Howse, J. W. S. Laidley, T. W. Lipscomb, I. Douglas Miller, J. S. MacMahon, Clarence Read, H. H. Schlink, J. C. Storey, Sir Robert Wade, C. E. Winston.

Queensland: E. D. Ahern, J. C. Hemsley, A. E. Lee, H. S. McLelland, Neville G. Sutton.

South Australia: A. M. Cudmore, H. M. Jay, I. B. Jose, Sir Henry Newland, T. G. Wilson.

Victoria: A. E. Brown, Sir Hugh Devine, T. E. Victor Hurley, A. Fay Maclure, J. Newman Morris, Sir Alan Newton, C. Gordon Shaw, Henry Searby, B. T. Zwar.

Western Australia: J. P. Ainslie, F. J. Clark, H. H. Stewart.

New Zealand: F. S. Batchelor, F. Gordon Bell, P. Stanley Foster, Sir Carrick Robertson, David Whyte, D. S. Wylie.

Admission of New Fellows.

The following new Fellows were admitted by the Council:

General Surgery.

New Zealand: Keith Buchanan Bridge, Alfred Bramwell Cook, Allan Augustus Macdonald.

Victoria: Howard Hadfield Eddey, John Gregory O'Donoghue.

Ophthalmology.

New South Wales: Edward Vanderbyl Waddy Pockley.